ADULT-PLANT RESISTANCE TO *PUCCINIA RECONDITA* f. sp. *TRITICI* IN A COLLECTION OF WILD *TRITICUM* SPECIES

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List of abbreviations

AP	appressorium
APR	adult-plant resistance
ASSV	aborted substomatal vesicle
ASSVI	aborted substomatal vesicle initial
C	chlorosis
CIMMYT	International Maize and Wheat Improvement Centre
cm	centimetre(s)
d	day(s)
d.p.i.	days post-inoculation
EA	early abortion
e.g.	*exempli gratia* (for example)
et al.	*et alii* (and others)
etc.	etcetera
Fig(s).	Figure(s)
f. sp.	*forma specialis*
G	germtube
g	gram(s)
H	haustorium
h	hour(s)
HCN	host cell necrosis
HI	hypersensitivity index
HMC	haustorium mother cell
HN	haustorium neck
h.p.i.	hours post-inoculation
HR	hypersensitive reaction
Hy	hypha
i.e.	*id est* (that is)
IH	infection hypha
IT	infection type
L	litre(s)
Lr	leaf rust resistance gene

m	metre
mg	milligram(s)
min.	minutes
ml	millilitre(s)
mm	milimetre(s)
mol	molar
MR	moderately resistant
MS	moderately susceptible
N	necrosis
NC	necrotic cell
NPA	nonpenetrating appressorium
NSA	nonstomatal appressorium
P	papilla(e)
PIH	primary infection hypha
R	resistant
St	stoma
S	susceptible
s	second
SIH	secondary infection hypha
ssp.	subspecies
SSV	substomatal vesicle
SSVI	substomatal vesicle initial
U	urediospore
var.	variety
V	vesicle
v	volume
w	weight
%	percentage
°C	degree Celsius
μ	micro
&	and
π	pi

Acknowledgements

To those who believe, there is
no failure.

Flavia Weedn

GENERAL INTRODUCTION

Rusts are conspicuous diseases of wheat affecting the foliage, stems and heads of plants. The pathogens causing these diseases are members of the fungus order Uredinales, which is synonymous with the term rust fungi. The three rust diseases of wheat belong to the genus *Puccinia* (Knott, 1989a). Leaf rust of wheat, caused by *P. recondita* f. sp. *tritici*, is an extremely serious disease worldwide and has been considered to account for the greatest losses in wheat due to cereal rusts over the long term (Wahl *et al.*, 1984). Although losses incurred may not be of the same magnitude as those of stem or stripe rust, yield reductions of up to 40% can occur (Knott, 1989a; Das *et al.*, 1992). The leaf rust fungus attacks a wide variety of hosts. However, there seems to be a strict specialisation of the various *formae specialis* towards host range. *P. recondita* f. sp. *tritici* is primarily a pathogen of wheat, its immediate ancestors and the man-made crop triticale (Roelfs *et al.*, 1992). To manage diseases caused by constantly changing rust pathogens, the need exists for an ongoing resistance breeding programme. According to McIntosh *et al.* (1995) the genetic base of common wheat is broadened by the identification and transfer of genes from relatives. Several examples of successful transfers exist (Knott & Dvorak, 1976; Fraunstein & Hammer, 1985; Gill *et al.*, 1985; Valkoun *et al.*, 1985; Valkoun *et al.*, 1986; Manisterski *et al.*, 1988; Singh *et al.*, 1988; Kerber & Dyck, 1990; Damania *et al.*, 1991; Dhaliwal *et al.*, 1991; Dhaliwal *et al.*, 1993; Dimov *et al.*, 1993; Dyck and Bartoš, 1994; Antonov & Marais, 1996). In wheat, these alien genes usually mediate race-specific, hypersensitive, and often non-durable resistance. Partial resistance (slow rusting) may be more durable than hypersensitive resistance and is expressed by a susceptible host reaction but slower rate of disease development. Components of resistance in slow rusting cultivars are longer latent periods, smaller and fewer uredia, and reduced spore production. Partial resistance has, however, not been studied extensively in wild wheats. Evidence also exists that resistance activated prior to haustorium formation may be more longlasting (Heath, 1981b; Heath, 1982; Heath, 1985). The objective of this study was to identify and characterise new sources of resistance to wheat leaf rust that could be exploited in future breeding.

CHAPTER 1

LITERATURE REVIEW

INTRODUCTION

Rusts are conspicuous diseases of wheat affecting the foliage, stems and heads of plants. Due to their wide distribution, genetic variability, effective long distance dissemination, and losses they cause, rusts are regarded as some of the most important diseases of wheat worldwide (Samborski, 1985; Schafer, 1987).

Historically, rusts were among the earliest recognised diseases of wheat. In the Bible, various references to rust diseases are found. Moses had warned the people that their crops will be destroyed by "smut and rust" if they do not obey the commandments of Jehovah (Deut.28:22). Also, the Samaritans were smitten "with smut and with rust" for oppressing the poor and crushing the needy (Amos 4:9) (Chester, 1946).

The growing of wheat in South Africa dates from shortly after Jan van Riebeeck settled in the Cape of Good Hope (presently Cape Town), in 1652. Considering the African continent, South Africa is the most important wheat producing area south of the equator (Payne et al., 1995). The wheat grown in South Africa is predominantly common or bread wheat (Triticum aestivum L.). The main factors determining the distribution of wheat production are the amount, seasonal distribution and consistency of rainfall. In most regions, rain occurs mainly in the summer months, when high temperatures are unfavourable for the development of the wheat plant, and this rain is, moreover, undependable (Peterson, 1965). More recently, economic factors such as the price of locally produced wheat, international competition and a free grain marketing strategy have also influenced wheat production in South Africa.

Theal indicated that rust was unknown in South Africa until 1727, but G.W. Thompson noted that severe rust epidemics occurred between 1708 and 1710 (Chester, 1946). According to Chester (1946) 1820 was a notorious rust year in South Africa.

HISTORY AND IMPORTANCE OF WHEAT RUSTS

Aristotle (384-322 B.C.) noted that the occurrence of rust epidemics differed from year to year and attributed this seasonal occurrence to variation in temperature and moisture. His student, Theophrastus Eresius (371-286 B.C.), noted particular susceptibility of cereals to rust, especially when the crops were grown in valleys or sheltered places. Columella (50 B.C.) burned piles of chaff in winter to avoid frost and rust injury (Chester, 1946).

From the year 1600 onwards there were numerous references to rust, although the various species of rust attacking wheat were not distinguished. Fontana recognised rust as a parasitic fungus on cereal plants in 1766, and was apparently the first to do so. Leaf rust of wheat was distinguished as a distinct species in 1815 by de Candolle when he described the causal organism as *Uredo rubigo-vera*. Prior to this event, the three rusts of wheat were collectively considered as a single disease (Chester, 1946).

Towards the end of the 19 th century the different rust species were classified separately. In 1894, Eriksson and Henning described *Puccinia dispersa* (Eriks. & Henn.), which included the leaf rusts of wheat and rye. Eriksson redescribed wheat leaf rust as *P. triticina* (Eriks.) in 1899 (Chester, 1946). Currently, the term *P. recondita* Rob. ex Desm. f. sp. *tritici* (Eriks. & Henn.) is accepted by most, if not all, leaf rust researchers (Samborski, 1984). Despite the common usage of the latter nomenclature, Anikster *et al.* (1997) provided evidence that the wheat pathogen should be renamed *P. triticina* as a separate species from the rye form.

De Bary described germination of urediospores in 1853, penetration through the stomata and development of uredia, as well as the germination of teliospores and production of urediospores. He further described germination of basidiospores on alternate hosts, development of appressoria, direct penetration of epidermal cells and formation of pycnia and aecia (Schafer *et al.*, 1984).

According to Schafer *et al.* (1984) Allen, from 1879 to 1963, also studied the histology of infection in and on cereal and alternate hosts. His work on uredial development was largely a comparison between resistant and susceptible wheats. His research had shown the collapse and death of host cells in resistant wheats and remains the basic reference concerning compatibility between cereal host and rust

pathogen (Schafer *et al.*, 1984).

Efforts to develop rust resistant wheat varieties were initiated in Kansas in 1911 (Chester, 1946). In 1915, McFadden crossed a resistant emmer wheat with Marquis and a cultivar named Hope was released. Hope was not a successful cultivar, but has probably been one of the widest used sources of stem rust resistance in the world (Schafer *et al.*, 1984).

Leaf rust of wheat, caused by *P. recondita* f. sp. *tritici*, is an extremely serious disease worldwide and has been considered to account for the greatest losses in wheat, among cereal rusts, over the long term (Wahl *et al.*, 1984). Its importance may vary from area to area according to climate and the degree of resistance in predominant cultivars. Year to year differentiation in an area depends primarily on the weather. Although losses incurred may not be of the same magnitude as those of stem or stripe rust, yield reductions of up to 40% can occur (Knott, 1989a; Das *et al.*, 1992).

THE WHEAT LEAF RUST PATHOGEN

Morphology, environmental requirements and symptoms The pathogens causing rust diseases of wheat are members of the fungus order Uredinales, which is synonymous with the term rust fungi. These are all obligate parasites on plants (Schafer, 1987). All three rusts belong to the genus *Puccinia*, but they differ in morphology, life cycle and environmental conditions required for optimal growth (Knott, 1989a).

Wheat leaf rust is caused by *P. recondita*. This is a complex species with considerable variation. The specialised form attacking wheat is *P. recondita* f. sp. *tritici* (Schafer, 1987). In comparing several characteristics of *P. recondita* worldwide, Anikster *et al.* (1997) concluded that two major groups could be distinguished within this complex. Isolates belonging to group I originated from species of cultivated wheats and wild emmer, whereas those in group II were collected principally from wild wheats and rye.

Leaf rust is the commonest and most widely distributed of the wheat rusts (Peterson, 1965; Schafer, 1987; Knott, 1989a). It occurs in all wheat growing regions of the world, but is most destructive in humid regions, and in moist seasons in the drier

regions. Leaf rust usually appears earlier in the growing season than stem rust, and thus has more time to multiply and reach epidemic proportions. It is favoured by warm, humid weather with frequent dews or showers (Peterson, 1965).

The spores of the leaf rust fungus are wind-borne (Peterson, 1965). Urediospores are 15-30 μm in diameter, subgloboid, with 3 to 8 germ pores scattered in their thick echinulate walls (Wiese, 1977). The pathogen primarily attacks leaf blades and to a lesser extent leaf sheaths, glumes and awns (Knott, 1989a). Leaf rust pustules are orange or brown, leading to the synonyms, brown or orange rust. The pustules are smaller (about 1-2 mm in diameter) than those of stem rust, and commonly oval-shaped or circular (Schafer, 1987; Knott, 1989a). These pustules are more numerous on the upper than the lower leaf surface and often become quite crowded. As the plant approaches maturity, black pustules may be formed in the tissue under the epidermis of the leaf or stem (Peterson, 1965).

Life cycle Wheat leaf rust is a macrocyclic rust with a sexual cycle on an alternate host and an asexual cycle on wheat (Fig. 1). Urediospores initiate germination 30 min. after contact with free water at temperatures ranging from 15 to 25°C. Few, if any, infections occur when dew period temperatures are above 32°C or below 2°C . The germtube grows along the leaf surface until it reaches a stoma, an appressorium is formed, followed by the development of a penetration peg and a substomatal vesicle from which primary hyphae develop. A haustorial mother cell develops against a mesophyll cell and direct penetration occurs. The haustorium is formed inside the living host cell in a compatible host-pathogen interaction. Secondary hyphae develop resulting in additional haustorial mother cells and haustoria (Roelfs *et al.*, 1992). After successful colonisation of the host, uredia with urediospores are formed under the epidermis on the adaxial sides of the leaves. In an incompatible host-pathogen interaction, haustoria fail to develop or develop at a slower rate (Rowell, 1981; Rowell, 1982), or the host cell containing the fungus dies. Depending on when or how many cells are involved, the host-pathogen interaction will result in a visible response (Roelfs *et al.*, 1992).

Spore germination to sporulation can occur within a seven to 10 day period at optimum temperatures. At lower temperatures (10-15°C) longer periods are necessary. Maximum sporulation is reached about four days after initial sporulation. The fungus

may survive as mycelia for a month or more when temperatures are near or below freezing (Roelfs *et al.*, 1992).

During unfavourable conditions or senescence, dicaryotic teliospores develop under the epidermis where they remain. Basidiospores are formed and released under humid conditions, which limit their spread. They are also hyaline and sensitive to light, which further limit dissemination (Roelfs *et al.*, 1992). After germination of a basidiospore and infection of the alternate host, the haploid mycelium produces pycnia with pycniospores. Through sexual fusion aecia with dicaryotic aeciospores are formed and released. The germination of aeciospores and infection of wheat result in dicaryotic mycelium. Finally, uredia with dicaryotic urediospores are formed (Nilsson, 1983).

Hosts The leaf rust fungus attacks a wide variety of hosts. However, there seems to be a strict specialisation of the various *formae specialis* towards host range. *Puccinia recondita* f. sp. *tritici* is primarily a pathogen of wheat, its immediate ancestors and the man-made crop triticale (Roelfs *et al.*, 1992).

Primary hosts The primary host of wheat leaf rust is *T. aestivum*. The disease has generally been of lesser importance on *T. turgidum* L. and of minor importance on *T. monococcum* L., *T. dicoccum* and *T. speltoides* (Tausch) Gren. *ex* K. Richt. (Roelfs *et al.*, 1992).

Alternate hosts In most areas (North America, South America, Australia and South Africa) the alternate hosts do not appear to play a major role, if any, in the life cycle. However, in some areas (Soviet Far East, Siberia, Japan), the sexual cycle is important in the production of new combinations of virulences by genetic recombination (Samborski, 1985). The alternate hosts for *P. recondita* are in the Ranunculaceace and Boraginaceae families. Several species of *Thalictrum*, *Anchusa*, *Clematis* and *Isopyrum fumarioides* can serve as alternate hosts (Roelfs *et al.*, 1992).

Accessory hosts *Puccinia recondita* attacks many species of grasses, but it is unclear which ones serve as functional hosts in nature for the *forma specialis tritici*. Potential hosts for wheat leaf rust could be wild or weedy species of the genera *Triticum* and *Aegilops* L. (now classified as *Triticum*) and the related species of *Agropyron* and *Secalis*. The most common non-crop host is volunteer or self-sown wheat. These

plants may be in fallow fields, along the edges of fields and roads, as weeds in a rotation or nearby crop, as a cover crop under orchards, along irrigation canals, etc. This is the major source of inoculum throughout much of the world where wheat is autumn- or winter sown (Roelfs *et al.*, 1992).

WHEAT

Origin and evolution All wheats, wild and cultivated, belong to the genus *Triticum* of the family Gramineae. The wheats (*Triticum* spp.) form a polyploid species with diploid (2*n*=2*x*=14), tetraploid (2*n*=4*x*=28) and hexaploid (2*n*=6*x*=42) forms (Miller, 1987; Knott, 1989a). The term wheat usually refers to the cultivated species of the genus *Triticum*. A number of species have been cultivated over the years, however, cultivation is now restricted almost entirely to the tetraploid durum wheat (*T. turgidum*) and the hexaploid common or bread wheat (*T. aestivum*) (Knott, 1989a).

Based on genome analysis, cultivated wheats evolved as shown in Figure 2 (Knott, 1989a). The various hexaploid wheats possess the A genome, probably originating from *T. boeoticum*, the B genome, probably derived from *Aegilops speltoides* Tausch Tausch or a related grass, and the D genome, which is thought to be derived from *Ae. squarrosa Coss.* (syn. *T. tauschii* (Coss.) Schmal.). It is believed that *T. spelta* evolved from a cross between *T. dicoccoides* or *T. dicoccum*, with the genomes AABB and *T. tauschii*, having the DD genomes. Cytological and genetical studies of *T. spelta* have indicated it to be the original form of cultivated hexaploid wheat. It is suggested that the mutation of one gene in *T. spelta* gave rise to *T. aestivum* (Peterson, 1965).

The origin of the B and G genomes has been the subject of considerable speculation and investigation and remains largely unresolved. Several possibilities exist for the origin of the B genome; the original donor may now be extinct, the donor may be a yet undiscovered diploid species, the genome may be derived from more than one source, or a rearrangement of the DNA may have occurred since its incorporation into the tetraploid (Miller, 1987).

Common wheat shares its AABB genomes with durum and the cultivated and wild emmers (*T. turgidum*, 2*n*=28). These tetraploids are relatively closely related to cultivated *T. timopheevii* (Zhuk.) Zhuk. and its wild relative, *T. araraticum* (AAGG).

Each of these species has contributed genetic variation to present day cultivated hexaploids. Likewise, diploid wheats (*T. monococcum*) and various diploid and polyploid relatives have acted as germplasm sources in wheat breeding (McIntosh *et al.*, 1995).

Classification of wheat Despite many years of research on evolutionary relationships in the wheat complex, the taxonomy of the wheats remains controversial. Traditionally, these taxa have been placed in either *Triticum* or *Aegilops*. More recently, they all have been grouped into one enlarged genus, *Triticum* (Morrison, 1993). Earlier, Peterson (1965) stated that the incorporation of *Aegilops* into the genus *Triticum* has not been universally accepted.

In 1753, Linnaus named seven genera in the tribe *Triticeae* including both *Triticum* and *Aegilops*. The genus *Triticum* contained species with cultivated forms and *Aegilops* the wild relatives. This classification was used by taxonomists for some 200 years (Kimber & Feldman, 1987). Morrison (1993) concluded that many of the wheat classifications in current use are inadequate or incomplete. Those that are cytogenetically based have a limited utility for researchers who must rely on other characters to identify and select germplasm.

One of the most readily available methods of classifying a wheat species is to compare the morphological characteristics of the wheat under study with those of other wheats and their relatives. The effectiveness of this method could be seen in the classification of Schultz in 1913. According to Peterson (1965), Schultz classified the known wheats into three groups (einkorn, emmer, and dinkel) (Table 1) on morphological grounds alone. Members within a group were related more closely than those in different groups.

Since that time, much research has left this group unchanged, except for the addition of new species, and the exclusion of *T. capitatum*. In 1921, Percival classified many forms of wheat on their morphological characteristics alone, and assigned them to their appropriate groups and species. Eig and Zhukovsky both called the non cultivated species *Aegilops* (Peterson, 1965). Kihara, Hammer and Gupta and Baum also separated the two genera, whereas, Stebbins had proposed that the two genera be amalgamated into one, since there was essentially no barriers between them

(Kimber & Feldman, 1987). Bowden proposed a classification including *Triticum* and *Aegilops* into one genus, *Triticum*. Morris and Sears adopted this classification unchanged (Kimber & Feldman, 1987).

Although there is still not complete agreement among taxonomists, many now include species formerly classified as *Aegilops* in the *Triticum* genus. Also, the former *Triticum* species having the same ploidy level, are consolidated into single species with the exception of *T. timopheevii* and *T. zhukovskyi* Menabde & Ericzjan which carry the G rather than the B genome. Within a ploidy level, all of the original *Triticum* species cross readily and produce fertile hybrids (Knott, 1989a).

The wheat group is characterised by a group of diploid species, in which there are eight distinct genomes (Table 2) and a group of polyploids (tetraploid and hexaploid) (Table 3) in which seven of the eight diploid genomes plus two more (B and G) are found (Kimber & Sears, 1987).

Wild relatives of wheat comprise two groups; the first group includes immediate progenitors of the cultivated wheats and the second those more distant relatives not directly involved in the evolution of wheat. The genetic variation in the former group is more readily available for use in wheat breeding. This group includes tetraploid wheats (*T. dicoccoides* [AABB] and *T. araraticum* [AAGG]); diploid wheats (*T. boeoticum* [AA] and *T. urartu* [AA]) and goatgrass (*T. tauschii* [DD]) (Gill *et al.*, 1986).

Relationships among species Hexaploid wheat (*T. aestivum*) exists primarily under cultivation and is reproductively isolated from the vast reservoir of gene pools contained in the diploid and tetraploid progenitor (A, D and the AABB genomes) species. Based on genomic affinities, the A, B and D genome progenitor species constitute the homologous gene pool. Other species that carry a different genome constitute the homoeologous gene pool. The related polyploid species that carry only one of the wheat genomes (eg. AAGG and CCDD species) constitute a partially homologous gene pool. Genes in the homologous pool can be transferred by chromosome pairing and crossing-over, whereas special and rather complex manipulations are often necessary for the transfer of genes from the homoeologous gene pool. Gene transfer from the homologous gene pool may be complicated by the sterility caused by differences in chromosome number, cross-compatibility barriers, especially between diploid species

and common wheat, complementary genes that cause seeding lethality and the general impairment of yield potential (Gill & Raupp, 1987).

The genus *Triticum* contains a broad range of species (Table 4), some of which cross readily with the cultivated tetraploid (*T. turgidum*) or hexaploid (*T. aestivum*) wheats, and others only with great difficulty. Wheat will also cross to some extent with species in a number of other genera including *Agropyron, Elymus, Hordeum* and *Secale* (Knott, 1987).

In the diploid group, *T. boeoticum* and *T. monococcum* can be readily crossed to produce fertile hybrids with seven pairs of homologous chromosomes. The two species have many heritable characters in common. The cytological and genetical evidence thus supports the morphological and physiological studies conducted earlier. It clearly indicates that the cultivated *T. monococcum* evolved from the wild species *T. boeoticum* (Peterson, 1965).

In most studies of genetic transfer from diploid progenitor species to wheat, the tetraploid wheat *T. turgidum* var. durum was used as a bridging species (Sharma & Gill, 1983). A triploid bridge can be used to introgress genes from *T. monococcum, Ae. speltoides* and *Ae. longissima* Schweinf. & Muschl. into durum wheat. From durum wheat, genes can then be transferred to hexaploid wheat (Kerber & Dyck, 1973). Durum wheat can also be used to transfer genes from *T. tauschii* by the formation of a synthetic hexaploid wheat. This approach has been successfully used to transfer several disease resistance genes from *T. tauschii* to hexaploid wheat (Kerber & Dyck, 1969).

Direct introgression from diploid species into hexaploid wheat is more difficult and may require specialised techniques. The crossability of wheat cultivars, F_1 seed abortion, F_1 hybrid lethality and high male and female sterility of F_1 hybrids are major hurdles (Gill & Raupp, 1987). In spite of the difficulties encountered, direct hybridisation allows rapid genetic transfer of useful traits to an adapted cultivar and can be a valuable applied technique (Gill & Raupp, 1987). *Triticum tauschii* may be the most suitable among the progenitor species for direct introgression. There is complete homology between hexaploid wheat D-genome chromosomes and those of *T. tauschii* (Riley & Chapman, 1960). The total genetic variation is more readily accessible, little adverse genetic interaction occurs between the D genome of wheat and that of *T. tauschii* and

there is evidence that *T. tauschii* has greater useful genetic variability than is found in the other progenitor species (Gill *et al.*, 1986). The use of embryo rescue facilitated direct genetic transfers from *T. tauschii* to hexaploid wheat and thereby averted the need of bridging species or prior synthesis of *T. turgidum* X *T. tauschii* amphiploids to overcome interspecific cross-incompatibilities. Several lines resistant to leaf rust were selected after direct genetic transfer from *T. tauschii* to hexaploid wheat (Gill & Raupp, 1987).

The wild tetraploid species, *T. dicoccoides*, is believed to be the original member of the tetraploid group. It is believed that *T. boeoticum* (AA) have crossed in nature with a diploid wild grass having a genome similar to the B genome of tetraploid wheat. This is believed to have been the ancestor of *Ae. speltoides* Tausch, which SS genome shows much homology with the BB genomes. The 14-chromosome hybrid would be sterile, as the chromosomes would not pair normally. Through fusion of reproductive cells containing 14 chromosomes with A and B genomes, the new wild species *T. dicoccoides* originated. This is believed to have occurred long before the domestication of wild wheats (Peterson, 1965).

Through mutation in *T. dicoccoides* and selection by farmers, the cultivated species, *T. dicoccum*, arose. This species provided far greater opportunities for mutation, natural crossing and selection, than did T. *dicoccoides*. The remaining tetraploids may therefore have evolved from *T. dicoccum* (Peterson, 1965).

The race or subspecies of *T. diccocoides* known as *T. araraticum* probably developed in the same manner but became isolated from the main stream of *T. dicoccoides* and developed genetic differences. Since *T. timopheevii* resemble *T. araraticum* in having the AA genomes and the modified BB genomes which are usually referred to as GG, it seems possible that *T. timopheevii* evolved from *T. araraticum* through mutation (Peterson, 1965).

The extent to which homoeology exists between the genomes within the *Triticeae*, such as those found in *Aegilops*, *Agropyron*, *Secale* and *Hordeum* species, which are potential donors of useful variation to wheat, is important in assessing the probable success of transfers from these species. In any wheat-alien exchange, even when efforts are made to ensure that the segment of chromosome transferred is as small as possible, genes other than at the initial target locus will be transferred. Close

homoeology should ensure that the wheat genes removed are replaced by similar genes from the alien donor, and, aided by the buffering already present in hexaploid wheat, larger segments may be tolerated. It is important that as few deleterious gene combinations as possible are included in such segments (Gale & Miller, 1987).

EARLY WHEAT BREEDING AND CONCEPTS OF RESISTANCE

Until late in the nineteenth century plant breeding appears to have been pursued more as an art than a science (Peterson, 1965). Previously, crops consisted of land races, which had evolved in the area where they were grown. Knight was the first to attempt cereal hybridisation. He grew contrasting varieties in close proximity and claimed to have produced superior varieties resistant to "blight" (Gale & Miller, 1987). The rediscovery of Mendel's work on inheritance in plants and the rapid advance in genetics and cytology also provided a strong impetus to the developing science of plant breeding. Thus, it has been particularly noteworthy in the twentieth century that wheat breeding, based on scientific principles and methods, has been greatly developed and extended (Peterson, 1965).

Ever since wild wheats were first domesticated, new forms have arisen from time to time through natural mutation and hybridisation. Forms that were better adapted to cultivation than the stock from which they arose, tended to survive. There can be no doubt, however, that some of the superior forms were recognised by farmers and consciously selected. This was an early form of wheat breeding (Peterson, 1965).

Until 1906, when the first wheat crosses were made in South Africa, wheat improvement depended mainly on introductions and selections. Most of these were too late maturing and lacked sufficient resistance to diseases. From about 1920 to 1930, earlier maturing wheat developed by hybridisation were predominant after which introductions became popular for a few years. From 1934 to 1958, Sterling wheat, developed by hybridisation, was the most dominant cultivar. Other important varieties of this period were Daeraad, Hoopvol and Impala. From 1957 onwards, Sokkies, an introduction from Kenya, became the most widely grown variety (Peterson, 1965).

It is the aim of the wheat breeder to develop wheats that are adapted to various environments, and have the yielding capacity and quality characteristics required by the

grower, processor and consumer (Peterson, 1965). High inherent yielding-capacity is the main objective in most breeding programmes. Varieties are usually bred to yield well under the soil and climatic conditions of a particular region (Peterson, 1965). Plant diseases are the most important yield limiting factor in wheat production and can contribute as much as 9.1% of losses (James, 1981). Yield reductions can be avoided by the use of resistant cultivars, but the leaf rust fungus is a very adaptable parasite and virulent pathotypes often develop soon after a resistant cultivar is released (Samborski, 1982). Selection pressure on the pathogen to develop virulence is particularly strong when a new resistant cultivar is grown on a large acreage. The lack of durability of resistance is mainly a problem with monogenic resistance to specialised pathogens, such as the rust fungi. Probable solutions to obtain durability are "pyramiding" of major resistance genes and race-nonspecific partial resistance (Niks & Rubiales, 1993).

Using flax rust (caused by *Melampsora lini* [Ehrenb.] Desmaz.) and its flax host (*Linum usitatissimum* L.), Flor demonstrated that if a cultivar carried a single gene for resistance, virulence in the pathogen was also conditioned by a single gene (Flor, 1942). Similarly, if resistance in a cultivar was conditioned by two genes, then virulence in the rust was conditioned by two genes. He stated: "These facts suggest that the pathogenic range of each physiologic race of the pathogen is conditioned by pairs of factors that are specific for each different resistant or immune factor possessed by the host variety". The significance of Flor's work was largely overlooked until Person (1959) published a theoretical analysis of gene-for-gene relationships in host-parasite systems. He concluded that such relationships should occur as a general rule in host:parasite systems as a result of selection pressures during evolution. Person *et al.* (1962) defined the gene-for-gene concept as follows: "A gene-for-gene relationship exists when the presence of a gene in one population is contingent on the continued presence of a gene in another population, and where the interaction between the two genes leads to a single phenotypic expression by which the presence or absence of the relevant gene in either organism may be recognised."

A gene for resistance to wheat rust has no selective advantage unless the pathogen carries the corresponding gene for avirulence, and a gene for virulence has no selective advantage if the host does not carry the corresponding gene for resistance. Most genetic analyses have shown that resistance to rust in wheat is controlled by

single dominant genes and virulence in the pathogens is controlled by corresponding recessive genes (Knott, 1989a).

Standard terminology for host-pathogen interactions was needed. Therefore, Loegering and Powers (1962) proposed that the character of the host be termed its reaction, which could either be resistant or susceptible; the character of the pathogen was named its pathogenicity, which could either be virulent or avirulent; and the interaction results in an infection type which may be low (resistance) or high (susceptibility).

The infection type descriptions used are based on the scale proposed by Roelfs (1988b). Following the original scale of Stakman *et al.* (1962) they developed a system of designating uredial infection types on a 0 to 4 scale with an extra class, designated X, for heterogeneous or mesothetic infections (Table 5). This system has been widely used for stem and leaf rust. Two additional classes are sometimes added, particularly for leaf rust. They are heterogeneous or pattern types. The Y infection type indicates variable uredium sizes with the largest most frequent at the tip of the leaf blade, whereas a Z infection type describes the more frequent occurrence of larger uredinia towards the base of the leaf (Knott, 1989a).

CHARACTERISATION OF RESISTANCE

Vanderplank (1963) proposed the existence of two different types of resistance in plants, viz. vertical and horizontal. He defined vertical resistance as being effective against some pathotypes and ineffective against others. It has therefore been called race-specific. In this type of resistance there is an interaction between genotypes of the host and pathogen. He defined horizontal resistance as being "evenly spread against all pathotypes of the pathogen". Therefore, no genetic interaction occurs between genotypes of the host and pathogen. This type of resistance thus is race-nonspecific.

Against each leaf rust pathogen, two types of resistance are recognizable: hypersensitive and partial resistance. Hypersensitive resistance is characterised by a low infection type, race-specificity and lack of durability (Parlevliet, 1988). Necrotic flecks may appear due to collapse of penetrated cells. This may be a complete or incomplete reaction (Parlevliet, 1981). Partial resistance is characterised by a reduced

rate of epidemic build-up despite a susceptible infection type, absence of large race-specific effects and durability. In the case of partial resistance no host cell collapse occurs (Parlevliet, 1981; Parlevliet, 1988).

The use of cultivars carrying hypersensitive resistance genes has been one of the most effective and economical means of controlling cereal rust (Nelson, 1978). Nearly all major gene resistances belong to this category (Parlevliet, 1988). The short-lived nature of hypersensitive resistance, however, has led to a search for alternative forms of resistance (Nelson, 1978). In this regard Sayre *et al.* (1998) concluded that the protection of yield potential in CIMMYT-derived spring wheats by the accumulation of genes conferring slow rusting has made a dramatic impact on wheat production.

The earliest studies pertinent to the hypersensitive reaction (HR) were directed at understanding the resistance of plants to obligate fungi. In the HR the disease is localised in the plant and the parasite is prevented from reproducing. The fungus enters both susceptible and resistant hosts in the same way but develops much different thereafter. In the susceptible host the fungus grows rapidly, without appearing to affect host cells for some time. In resistant hosts a rapid reaction develops resulting in the almost intermediate death of some host cells (Goodman & Novacky, 1994). According to Goodman & Novacky (1994), in 1915, Stakman observed that the more resistant a cultivar, the more rapid the death of a limited number of cells in the vicinity of the invading hyphae. The time course recorded for symptoms of the HR in resistant tissues is 2-3.5 days and for abundant spore production in susceptible tissue, 7 - 12 days (Goodman & Novacky, 1994).

Infection in resistant cultivars is not always associated with the development of extensive necrotic tissue. In 1902, Ward mentioned a condition of incompatibility in which no haustoria are formed, even though germination and penetration occur. This is a form of resistance where no necrosis develops. Although it was initially inferred that HR resulted in localisation and death of the pathogen in incompatible interactions, excision of hyphae from the HR milieu revealed them to be pathogenically competent in susceptible tissue (Sharp & Emge, 1958). By raising the incubation temperature, the resistant reaction apparently changed to a susceptible one. This suggested that hypersensitive necrosis does not kill the fungus (Silverman, 1959; Zimmer & Schafer, 1961). Brown *et al.* (1966) concluded that necrotic tissue in resistant hosts reveals

those hosts that are more sensitive to the disturbances caused by the fungus and that necrosis may be the consequence rather than the cause of resistance. Heath (1976), however, indicated that hypersensitivity may play a different role in different resistant responses. It is therefore necessary to look at each individual system from as many angles as possible. Brown et al. (1966) also emphasised that infection in resistant hosts is not always associated with necrosis.

Clearly, the HR, with its characteristic rapid cell death and subsequent necrosis, constitutes one of the primary mechanisms of resistance to plant disease. Whereas other mechanisms such as the development of papillae, callose, phytoalexins, cuticle, suberin and lignin all involve the synthesis of a cell-protecting entity, the HR, on the other hand, requires rapid host cell death (Goodman & Novacky, 1994).

Race-specific resistance of wheat to leaf rust is often short-lived whereas slow rusting has been reported to be a more durable type of resistance (Ohm & Shaner, 1976, Kuhn et al., 1978, Das et al., 1992). Slow rusting is a quantitative form of resistance where a susceptible host reaction is observed but the rate of disease development is restricted when compared to susceptible cultivars. Due to the functioning of several components of resistance, Kulkarni & Chopra (1980) considered slow rusting as the product of an interaction between the host and pathogen at different stages of pathogenesis. Partial resistance is not identical to slow rusting, as all incomplete resistance to rusts results in slow-rusting, including resistance with intermediate infection types (Parlevliet, 1988). Partial resistance is often recessive and the result of several genes with small to intermediate effects (Parlevliet, 1993). This type of resistance to leaf rust may be more durable than high levels of hypersensitive resistance (Lehman & Shaner, 1996).

Durable resistance is resistance which has been adequate against the disease for a number of years over a range of environments and pathogen cultures. According to Johnson (1981), disease resistance can only be classified as durable if cultivars possessing the resistance are widely cultivated. Cultivars with durable resistance retain their resistance despite large-scale, long-term exposure to the pathogen under conditions favourable for disease development (Wolfe, 1993). It should not be assumed that it will always be adequate, nor that it will be effective against all pathotypes. However, the use of resistance effective over a range of environments,

pathotypes and years, is more likely to lead to a resistant cultivar than resistance that is known to have failed. There are several known sources of durable resistance to stem rust which are related to the *Sr2* gene, while, for leaf rust, most durable resistance is associated with gene combinations (Roelfs, 1988a).

It has been argued that it is more difficult to obtain durable resistance to leaf rust than to stem rust (Roelfs, 1988a). Leaf rust is more diverse for virulence than stem rust. This could be attributed to several factors (Roelfs, 1988a). The population that survives between wheat crops probably is much larger for leaf rust, the pathogen population size is much larger during the crop season and resistance against leaf rust has often been a single gene at a time. Due to large populations, a greater probability of mutants exist, as well as a greater diversity of virulence/avirulence combinations can survive the non-wheat growing period (Schafer & Roelfs, 1985). Changes in virulence in leaf rust have been frequent (Samborski, 1982; Statler *et al.*, 1982; Bennett, 1984; Pretorius, 1988) and the number of usable genes is limited (Browder, 1980; Bennett, 1984).

At present there is little evidence documenting the durability of partial resistance (Lehman & Shaner, 1996). Furthermore, durable resistance is only identified retrospectively and more information on strategies to obtain longlasting resistance in directed breeding is needed.

COMPONENTS OF RESISTANCE

Many studies have characterised the resistance in cereal hosts to their respective rust pathogens. Macroscopic components of resistance are considered those that can be measured at the whole plant level to describe disease development, e.g. latent period, uredium density and size, and sporulation capacity. Several studies have ascribed the reduced rate of epidemic progress to a reduced infection frequency, longer latent period and reduced rate of spore production (Caldwell *et al.*, 1970; Gavinlertvatana & Wilcoxson, 1978; Parlevliet, 1979; Shaner & Finney, 1980; Lee & Shaner, 1985b; Parlevliet, 1985; Pretorius *et al.*, 1987a; Pretorius *et al.*, 1987b; Brière & Kushalappa, 1995). Latent period has been identified as one of the most important components of slow-rusting resistance (Parlevliet, 1975a; Ohm & Shaner, 1976; Shaner *et al.*, 1978).

Microscopic components, on the other hand, comprise detailed microscopy aimed at the characterisation of resistance expression at the cellular level. Studies have included partial resistance (Niks, 1981; Niks, 1982; Niks, 1983a), monogenic or digenic resistance (Sawhney *et al.*, 1992; Bender *et al.*, 1997; Jacobs *et al.*, 1996; Kloppers & Pretorius, 1997) and attempts to distinguish the onset of resistance in relation to haustorium formation (Niks, 1986; Niks & Dekens, 1991). Should high levels of prehaustorial resistance be identified, the assumption is that it could emulate a nonhost reaction, and thus a more durable type of resistance (Niks & Dekens, 1991).

BREEDING FOR RESISTANCE

In breeding for resistance to the rust diseases of wheat, the main objective is to develop cultivars that will remain resistant for at least the period when they are grown commercially (Knott, 1989a). Due to different breeding objectives and variation in factors such as the host, pathogen and environment, many approaches could be followed. According to Knott (1989a) the main breeding procedures include the pedigree and bulk systems, backcrossing, generations advanced by single seed descent, and recurrent selection. Selection for resistance during the various cycles of breeding is usually conducted in carefully planned field nurseries where rust epidemics are created artificially, or in controlled environments. To accelerate progress in e.g. the pedigree system, breeders often grow an off-season nursery which allows two generations to be advanced per year.

Considerable progress in leaf rust resistance breeding has recently been made in CIMMYT's wheat programme (Sayre *et al.*, 1998). Their breeding methodology is tailored to develop widely adapted, disease resistant germplasm with high and stable yields across a wide range of environments. The incorporation of durable, non-specific disease resistance in spring wheats has been a high priority since widely adapted germplasm would not have stable yields without adequate resistance against the major diseases. Intentionally, diverse sources of resistance to rust diseases are used. CIMMYT's strategy for resistance to cereal rusts is based on general resistance (slow rusting) (Braun *et al.*, 1996). Cultivars carrying slow rusting resistance show high infection types in the seedling growth stage (Singh, 1997). This non-specific resistance

can be further diversified by accumulating several minor genes and combine them with different specific genes to provide genetic diversity. About 60% of the CIMMYT germplasm carry one to four genes for partial resistance genes, including *Lr34* (Braun *et al.*, 1996).

Considering the utilisation of major gene resistance in wheat breeding, several alien genes for controlling resistance to wheat leaf rust have been transferred through wide hybridisation from wild progenitors and related species and genera (Dvórak, 1977; Sharma & Gill, 1983; Gale & Miller, 1987; Gill & Raupp, 1987; McIntosh *et al.* 1995; Friebe *et al.*, 1996), many of which have been exploited commercially (Sharma & Gill, 1983; Knott, 1989a). Depending on the genetic diversity of relatives, transferring alien genes can either be a simple or very complicated procedure (Knott, 1989b). Keeping in mind the variability for disease resistance and the ease of transfer, the choice of the donor species for the improvement of the cultivated wheats may be restricted to the more closely related species (Dhaliwal *et al.*, 1993).

Considering a molecular approach to wheat improvement, the isolation of race-specific resistance genes from cereals and avirulence genes from the pathogen offer new opportunities (Bushnell *et al.*, 1998). For cereals, the bombardment of tissues with microparticles coated with plasmid DNA is the most widely used method. Genetic material used for bombardment can include: (i) genes for disease resistance, (ii) defence response genes, (iii) genes related to pathogenicity, (iv) genes for antifungal proteins and (v) genes related to race-specific resistance. Transgenes should be utilised to minimise the ability of pathogen populations to overcome them, whereas for durability they should be designed to produce products at infection sites, and be tailored for individual plant diseases. Each transgene used in genetic engineering needs to be used together with other genes and in combination with other disease control practices (Bushnell *et al.*, 1998).

WILD RELATIVES OF WHEAT AS SOURCES OF RESISTANCE

The wild relatives of wheat have been shown to be a rich reservoir of resistance genes to leaf rust. High levels of resistance have been identified in several species e.g. *T. monococcum* (Kerber & Dyck, 1973; Valkoun *et al.*, 1986; Valkoun & Mamluk, 1993;

Dyck & Bartoš, 1994), *T. cylindricum* (Host) Ces., Pass. & Gibelli (Bai *et al.*, 1995), *T. tauschii* (Gill *et al.*, 1985; Gill *et al.*, 1986; Gill & Raupp, 1987; Cox *et al.*, 1992a), *T. speltoides* (Dvorak, 1977; Manisterski *et al.*, 1988), *T. triaristatum* (Bai *et al.*, 1993), *T. peregrinum* (Antonov & Marais, 1996) and *T. timopheevii* (Knott & Dvorak, 1976; Brown-Guedira *et al.*, 1996; Brown-Guedira *et al.*, 1997).

The catalogued *Lr* genes that have been transferred from wild relatives are *Lr9* (*T. umbellulatum* (Zhuk.) Bowden), *Lr14a* (*T. dicoccoides*), *Lr18* (*T. timopheevii*), *Lr19* (*Thinopyrum distichum*), *Lr21, 22a, 32, 39, 40, 41, 42, 43* (*T. tauschii*), *Lr23* (*T. turgidum* var. durum), *Lr24, 29* (*Th. ponticum*), *Lr25, 26, 45 (Secale cereale), Lr28, 35, 36* (*T. speltoides*), *Lr37* (*T. ventricosum*), *Lr38* (*Th. intermedium*) and *Lr44* (*T. spelta*) (McIntosh *et al.*, 1995; McIntosh, 1988).

The procedures for transferring genes to wheat from its more distant relatives work best with major genes which effects are easy to measure. Some resistances may be polygenic and difficult to transfer (Knott, 1989a). The successful transfer of single genes to wheat from alien species requires that the gene or a segment of chromosome carrying it can be incorporated into a wheat chromosome, that it is expressed in a similar way in the wheat genomes than in the alien species, and that any loss of wheat genetic material does not result in a wheat genotype inferior to the original (Gale & Miller, 1987).

When work on transfer of rust resistance from more distant relatives of wheat was started, it was hoped that this resistance might prove to be durable. There is, however, little evidence that this resistance differ from that in common wheat. Resistance from an alien source is often initially effective against a wide range of rust pathotypes. In a number of cases resistance from alien species have, however, been overcome by a new, virulent pathotype (Knott, 1989a; McIntosh *et al.*, 1995).

Many species of *Triticum* and related genera are cross-compatible. The genotypes of the parents are important in determining the success of a cross. In crosses between durum or bread wheat and close relatives, viable seeds are often produced. In wider crosses, embryo rescue might be necessary (Knott, 1989a).

Diploid wheats (*T. monococcum*) are a non-host to the wheat leaf rust fungus, *P. recondita* f. sp. *tritici* (The, 1976; Niks & Dekens, 1991). Over 99% of the accessions are resistant, often totally without symptoms. Histological studies showed

that the mechanism of resistance in diploid wheat to wheat leaf rust could be either pre-
or posthaustorial (Niks & Dekens, 1991). Segregation ratios suggested that
prehaustorial resistance is controlled by one recessive major gene and posthaustorial
resistance by one dominant major gene (Zhang *et al.*, 1993).

Cultivated diploid einkorn wheat, *T. monococcum*, has been recognised as a
valuable source of disease resistance genes. The high chromosome homology
between *T. monococcum* and durum and bread wheat, allows transfer of resistance
genes, without deterioration of agronomic characters (Valkoun & Mamluk, 1993). Until
recently, *T. monococcum*, has not been successfully used in wide hybridisation. In the
past two decades, however, genes for rust resistance have been transferred from *T.
monococcum* to hexaploid bread wheat (Kerber & Dyck, 1973; Valkoun *et al.*, 1986;
Hussien *et al.*, 1997; Hussien *et al.*, 1998) as well as from an autotetraploid of *T.
monococcum* (Dyck & Bartoš, 1994).

Bai *et al.* (1995) reported that three accessions of *T. cylindricum* were resistant
to several pathotypes of stem and leaf rust. Viable F_1 plants were produced from the
crosses between *T. cylindricum* and susceptible hexaploid wheats but not with
susceptible tetraploid durum wheat. Dimov *et al.* (1993) detected no resistance in
accessions of *Ae. cylindrica* Host.

Triticum tauschii is another valuable source of genes for diversifying pest
resistance in wheat. High levels of leaf rust resistance were obtained in accessions of
T. tauschii (Gill *et al.*, 1986; Cox *et al.*, 1992b). Because of the richness of genetic
diversity in *T. tauschii* for disease and insect resistance, it is mandatory that
conservation and utilisation of this genetic resource receive the highest priority (Gill *et
al.*, 1986).

The timopheevii wheats include the cultivated *T. timopheevii* var. timopheevii and
its wild progenitor *T. timopheevii* var. araraticum (Brown-Guedira *et al.*, 1997). Although
T. timopheevii probably has more disease resistance than any of the other *Triticum*
species (Brown-Guedira *et al.*, 1996), it has been used to a lesser degree.
Consequently, there has been considerable interest in using the timopheevii wheats as
a source of resistance (Knott & Dvorak, 1976). Cultivated *T. timopheevii* has been used
for wheat improvement to a greater extent than has its wild progenitor, *T. araraticum*.
Because *T. timopheevii* is a cultivated species, the chances of recovering agronomically

acceptable derivatives from crosses with wheat are greater. However, *T. araraticum* is found in diverse ecological regions, therefore, chances are greater of it containing more diverse useful genes for wheat improvement (Brown-Guedira *et al.*, 1997). Many problems are, however, faced when attempting to introgress genes from *T. araraticum* into wheat. Viable hybrid seed can be recovered from crosses between *T. araraticum* and hexaploid wheat without embryo rescue, but mature F_1 plants are sterile. Due to reduced recombination between *T. aestivum* and *T. araraticum*, recovery of desirable plant types in the progeny of the interspecific cross may be difficult (Brown-Guedira *et al.*, 1997).

Triticum dicoccoides does not have high levels of leaf rust resistance either as seedlings (Nevo, 1993; The *et al.*, 1993) or adult plants. Moseman *et al.* (1985), however, found 14% of a wild emmer collection to be resistant or moderately resistant to a race of leaf rust in seedling tests and Dyck (1994) transferred two genes from *T. turgidum* ssp. *dicoccoides* (Körn. *ex* Aschers. & Graebner) Thell. to hexaploid wheat.

Tetraploid species of *Triticum*, including *T. durum*, have been used as sources of rust resistance to provide genetic diversity in hexaploid cultivated wheat. Fifty accessions of *T. durum* germplasm were tested for resistance to leaf rust. In 43 of the accessions, reaction patterns could not be matched to a known *Lr* gene or a combination of *Lr* genes. This suggested that there is a large diversity for leaf rust resistance in this germplasm (Singh *et al.*, 1992). Leaf rust resistance have been successfully transferred from the durum wheat cultivars Medora and Stewart to hexaploid wheat (Dyck & Bartoš, 1994).

Antonov & Marais (1996) have shown that a rich and accessible source of new resistance genes exists in related species. They screened 877 *Triticum* accessions for resistance to leaf rust. Of these, 206 accessions were resistant or moderately resistant to the pathotypes used. Similarly, resistance to leaf rust was detected in 58% of an Ethiopian wheat collection consisting of tetraploid and hexaploid species (Negassa, 1987).

Sharma & Knott (1966) transferred leaf rust resistance from Agrus, a wheat-*Agropyron elongatum* derivative, to Thatcher. One of the resulting lines, later named Agatha, was used to produce leaf and stem rust resistant Thatcher backcrosses. Although they were all agronomically satisfactory, they had a distinctly yellow flour,

presumably conditioned by a gene on the translocated segment of the *Agropyron* chromosome.

Wild relatives of wheat needs to be screened extensively before useful genes can be fully utilised. When species carrying useful genes have been identified, the next step involve the transfer of useful genes to wheat. The success of the transfer depends on several factors: the difficulty in making the cross, the amount of pairing that occurs between the alien chromosome and the wheat chromosome (A, B, or D) and the genetic complexity of the character. On the basis of chromosome pairing, transfers from alien species to wheat can be divided into two categories: transfers between homologous chromosomes and transfers between nonhomologous, but often homoeologous chromosomes. In a species with one genome homologous to a wheat genome and one or more homoeologous chromosomes, the transfer can be in either category, depending on which genome carries the gene of interest. If several genes are involved, it is possible that both homologous and homoeologous transfers will be required (Knott, 1987).

The transfer of characters from one species or genus to another is not only of practical importance, but of considerable genetic interest as well. The greater the distance over which the transfers can be made, the greater the possibility of introducing useful characters not present in the host species. It is therefore important to extend the limits of transfer as far as possible (Sears, 1956).

CONCLUSIONS

Ongoing research in cereal rust genetics is necessary if losses from leaf rust of wheat are to be minimised. It has often been shown that cultivars with a single gene for resistance do not remain resistant for very long. Although resistance genes can be transferred from several wild relatives, using a variety of techniques, genes from such sources are not necessarily durable. Matching virulences to most of the alien genes for leaf rust resistance transferred to wheat have evolved (Dhaliwal *et al.*, 1993). In North America, virulent pathotypes of leaf rust appeared quickly after the release of cultivars with *Lr9* from *T. umbellulatum* and *Lr24* from *Th. ponticum*. Despite the occurrence of such virulence, careful gene management should provide sustainable control of leaf

rust. This can be achieved by the use of multilines and by the cultivation of cultivars with different genes for resistance (Samborski, 1984). In future, more emphasis should be placed on following the approach whereby slow rusting resistance is accumulated. In this regard effective alien genes, protected by several nonspecific genes, should prolong the lifespan of the former considerably.

The improvement of crossing techniques has impacted significantly on alien gene transfer from distantly related species. Crosses between wheat and any of the species in the Triticeae and species such as maize, sorghum are possible. However, posthybridisation barriers such as chromosome elimination, preferential transmission of certian alien chromosomes, and adverse genetic interactions leading to hybrid dysgenesis, chromosome breakage, and sterility, impede further progress in alien transfer. Diverse selection of host and donor genotypes in the initial hybridisation can often overcome some of these barriers (Jiang *et al.*, 1994).

The continuing need of wide hybridisation is supported by various arguments: (i) land races and wild species will continue to be reservoirs of genetic diversity and wide hybridisation is the best means to utilise this variation; (ii) wide hybridisation and production of addition and translocation lines are necessary steps for genetic characterisation of the alien phenotypic traits, and (iii) in a polyploid crop like wheat, transfer of adaptive linkage blocks may be more desirable than single gene transfers (Jiang *et al., 1994).*

Table 1: Classification of wheat into morphological groups (Peterson, 1965)

I. Einkorn Series	*T. aegilopoides* (*T. boeoticum*)
	T. monococcum L.
II. Emmer Series	*T. dicoccoides*
	T. dicoccum
	T. durum
	T. turgidum L.
	T. polonicum
III. Dinkel Series	*T. spelta*
	T. compactum
	T. vulgare
	T. capitatum (from *T. compactum* x *T. vulgare*)

Table 2. Proposed genome symbols for the diploid species of genus *Triticum* (Kimber & Sears, 1987)

Species	Symbol	Synonyms
T. monococcum L.	A	*T. boeoticum*
T. speltoides (Tausch) Gren. *ex* K. Richt.	S	*Aegilops speltoides* Tausch
T. bicorne Forssk.	S^b	*Ae. bicornis* (Forssk.) Jaub. & Spach
T. longissimum (Schweinf. & Muschl.) Bowden	S^l	*Ae. longissima* Schweinf. & Muschl., *Ae. sharonense* Eig
T. searsii (Feldman & Kislev) Feldman	S^s	*Ae. searsii* Feldman & Kislev *ex* K. Hammer
T. tripsacoides (Jaub. & Spach.) Bowden	M^t	*Ae. mutica*
T. tauschii (Coss.) Schmal	D	*Ae. squarrosa*
T. comosum (Sm. *in* Sibth. & Sm.) K. Richt	M	*Ae. comosa* Sm. *in* Sibth. & Sm., *Ae. heldrechii*
T. uniaristatum (Vis.) K. Richt.	U^n	*Ae. uniaristata*
T. dichasians (Zhuk.) Bowden	C	*Ae. caudata* L.
T. umbellulatum (Zhuk.) Bowden	U	*Ae. umbellulata* Zhuk.

Table 3. Proposed genome symbols for the polyploid species of the genus *Triticum* (Kimber & Sears, 1987)

Species	Symbol	Synonyms
T. turgidum L.	AB	*T. carthlicum, T. dicoccoides, T. dicoccon, T. dicoccum, T. durum, T. polonicum*
T. timopheevii L.	AG	*T. araraticum*
T. zhukovskyi Menabde & Ericzjan	AAG	*T. timopheevii* var. zhukovskyi
T. aestivum L.	ABD	*T. compactum, T. macha, T. spelta, T. sphaerococcum, T. vavilovii*
T. ventricosum (Tausch) Ces., Pass. & Gilelli	DUn *	*Aegilops ventricosa* Tausch
T. crassum (Boiss.) Aitch. & Hemsl. (4x)	DM	*Ae. crassa* Boiss.
T. crassum (Boiss.) Aitch. & Hemsl. (6x)	DDM	*Ae. crassa*
T. syriacum Bowden	DMS	*Ae. crassa* ssp. vavilovii, *Ae. vavilovii* (Zhuk.) Chennav.
T. juvenale Thell.	DMU	*Ae. juvenalis* (Thell.) Eig
T. kotschyi (Boiss.) Bowden	US	*Ae. kotschyi* Boiss., *Ae. peregrina* (Hackel *in* J. Fraser) Maire & Weiller, *Ae. variabilis*
T. ovatum (L.) Raspail	UM	*Ae. ovata*
T. triaristatum (4x)	UM	*Ae. triaristata*
T. triaristatum (6x)	UMUn	*Ae. triaristata*
T. macrochaetum (Shuttlew. & A. Huet *ex* Duval-Jouve) K. Richt	UM	*Ae. biuncialis* Vis., *Ae. lorentii*
T. columnare (Zhuk.) Morris & Sears	UM	*Ae. columnaris* Zhuk.
T. triuncale (L.) Raspail	UC	*Ae. triuncalis* L.
T. cylindricum (Host) Ces., Pass. & Gibelli	CD	*Ae. cylindrica* Host

Table 4. The relatives of wheat grouped according to their genomes and the presumed closeness of their relationship to bread wheat (Kimber & Sears, 1987)

Group	Species
1. Species carrying only the A, B, or D genomes.	
(a) The diploid progenitors	*T. monococcum*
	T. tauschii
(b) The tetraploid progenitor	*T. turgidum*
2. Polyploids with one homologous genome	
(a) The A genome	*T. timopheevii*
(b) The D genome	*T. cylindricum*
	T. ventricosum
	T. crassum
	T. syriacum
	T. juvenale
3. Species with only homoeologous genomes	
(a) Closely related species	*T. speltoides*
	T. bicorne
	T. longissimum
	T. searsii
	T. kotschyi
(b) Less closely related species	*T. dichasians*
	T. comosum
	T. tripsacoides
	T. uniaristatum
	T. umbellulatum
	Other U-containing polyploids
	Several *Elytrigia* species
(c) Distantly related species	Species of *Secale, Haynaldia, Hordeum, Agropyron, Elytrigia*, etc.

Table 5. Major infection type classes for stem and leaf rust (Roelfs, 1988b; McIntosh et al., 1995)

Infection type[a]	Host response	Symptoms
0	Immune	No visible uredia
;	Very resistant	Hypersensitive flecks
1	Resistant	Small uredia with necrosis
2	Resistant to moderately resistant	Small to medium sized uredia with chlorosis or necrosis
3	Moderately resistant/moderately susceptible	Medium sized uredia with or without necrosis
4	Susceptible	Large uredia without chlorosis or necrosis
X	Resistant	Heterogeneous, similarly distributed over the leaves
Y	Resistant	Variable size with larger uredia towards the tip
Z	Resistant[b]	Variable size with larger uredia towards the leaf base

[a] Infection types are often refined by modifying characters as follows: --, uredia at lower size limit; -, uredia somewhat smaller than normal; +, uredia somewhat larger than normal; ++, uredia at the upper size limit; C, more chlorosis than normal; and N, more necrosis than normal for the infection type.

[b] High leaf rust severities associated with a Z pattern may be regarded as susceptibility.
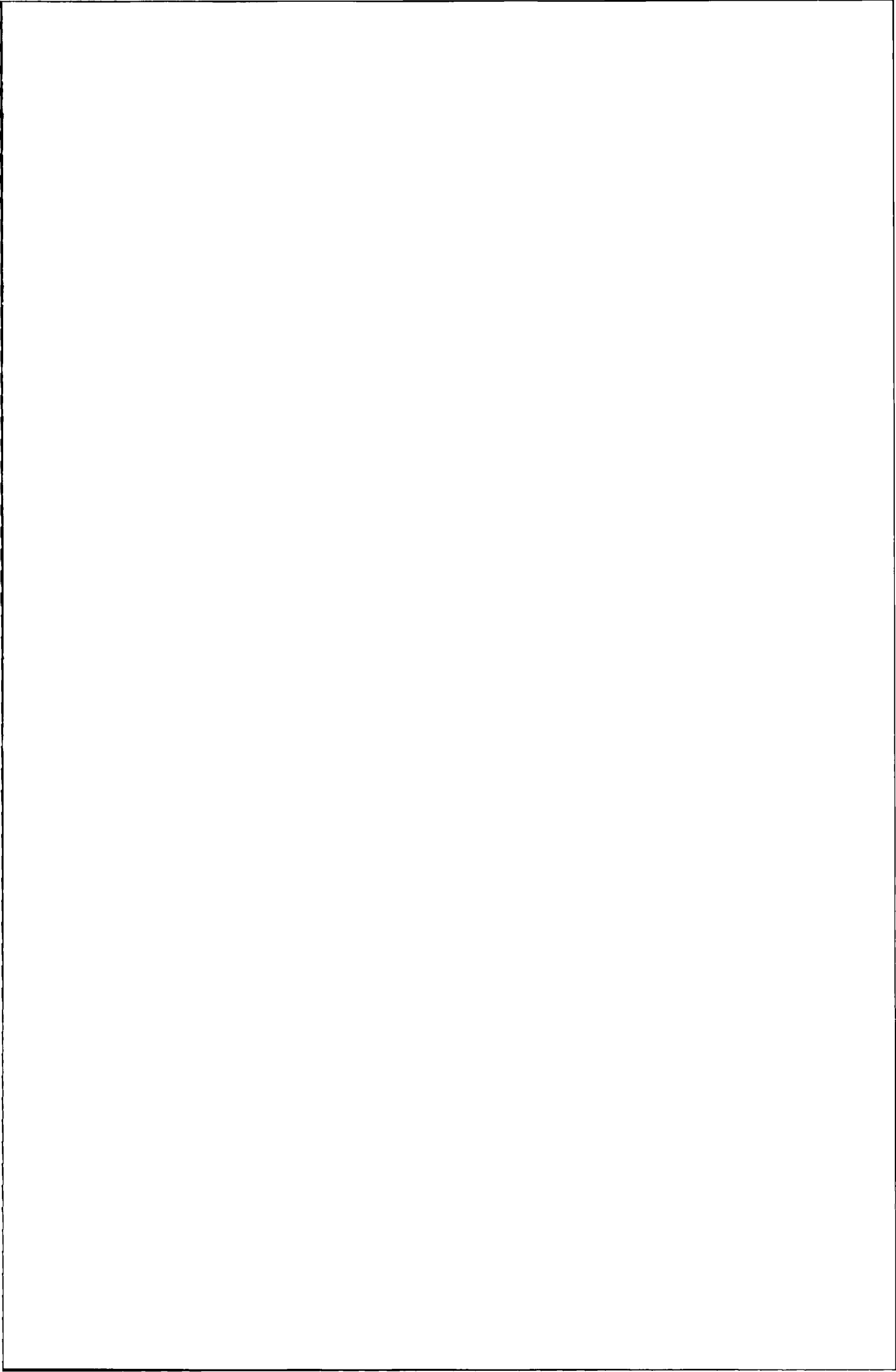
Figure 1. Life cycle of *Puccinia recondita* f. sp. *tritici* (wheat leaf rust) (Roelfs *et al.*, 1992).

Pycnia

Aeciospores

Uredinia on leaf

Aecia on *Thalictrum*

Telia on leaf

Pycnia on *Thalictrum*

Asexual cycle on wheat

Basidiospores on basidium

Basidiospores

Urediniospores

Spring

Summer

Winter

Tellospores
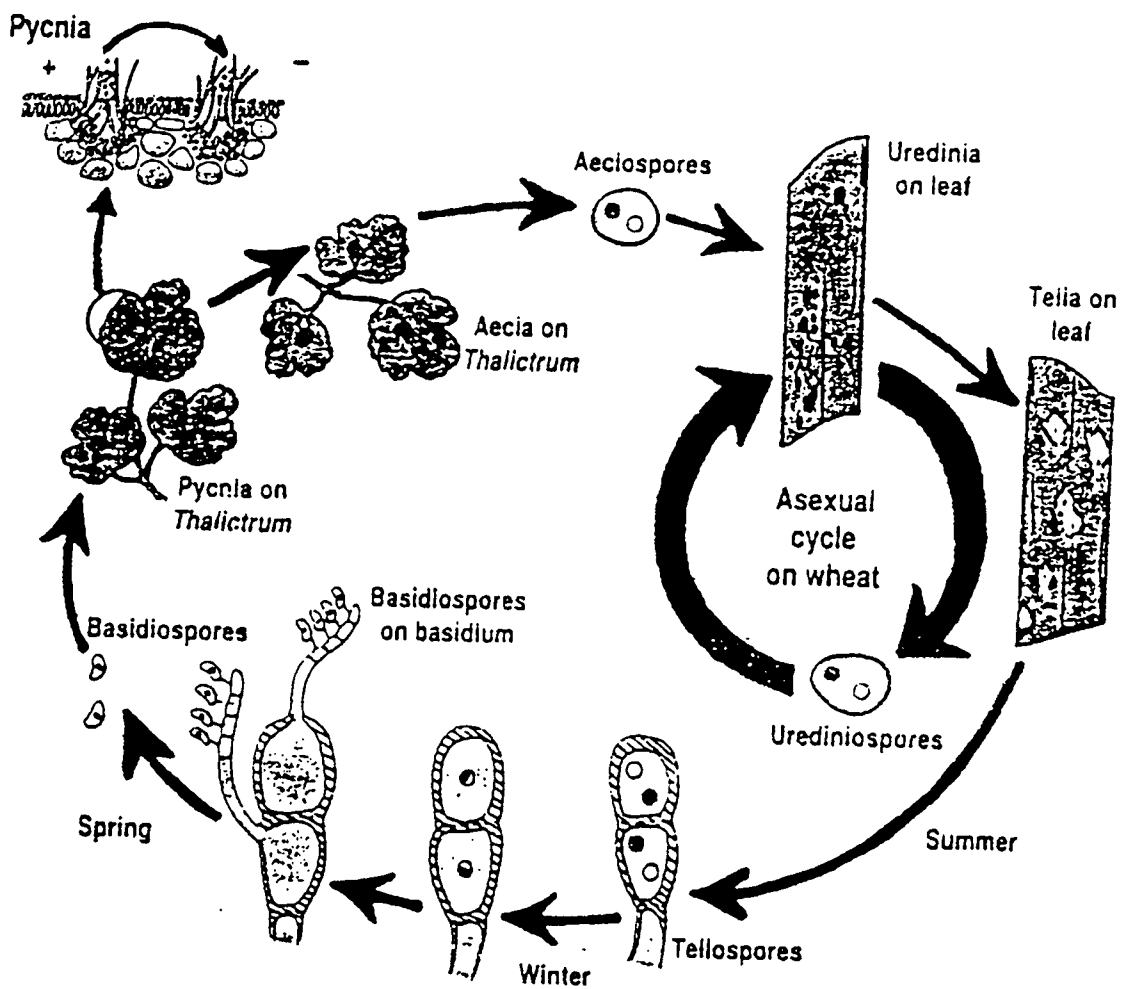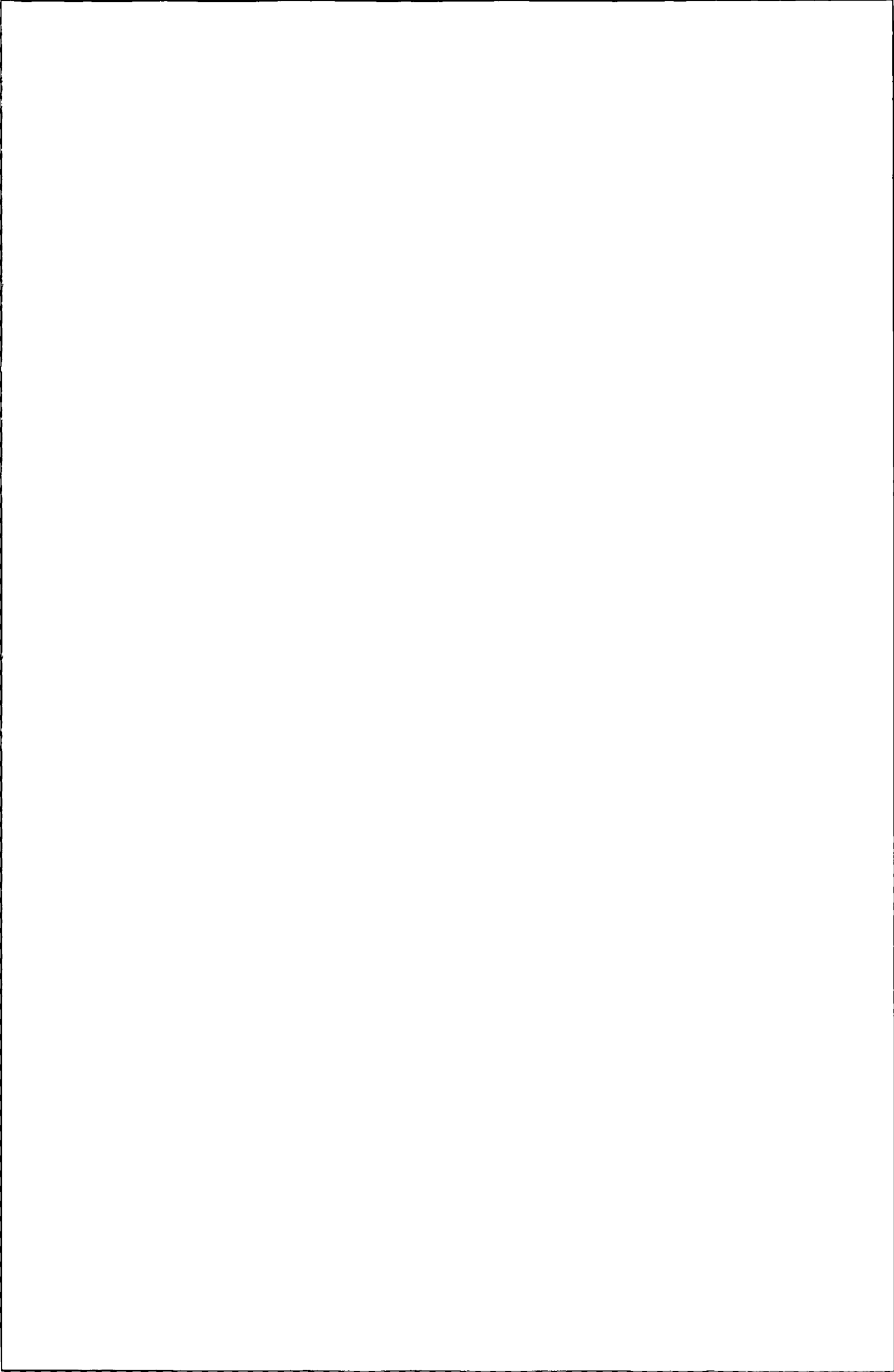
Figure 2. The origin of *Triticum turgidum* (durum wheat) and *Triticum aestivum* (bread wheat) (Knott, 1989a).

T. monococcum   x   Unknown species

(2n = 14, AA)   (2n = 14, BB)

↓

T. turgidum   x   *T. tauschii*

(2n = 28, AABB)   (2n = 14, DD)

↓

T. aestivum

(2n = 42, AABBDD)

CHAPTER 2

IDENTIFICATION OF WHEAT LEAF RUST RESISTANCE IN A COLLECTION OF WILD *TRITICUM* SPECIES

ABSTRACT

In an attempt to identify new sources of resistance to *Puccinia recondita* f. sp. *tritici*, 353 *Triticum* accessions, comprising 13 diploid, tetraploid or hexaploid species, were screened for seedling and adult-plant resistance to wheat leaf rust using a mixture of pathotypes UVPrt2, 3, 9 and 13. Seedlings were spray-inoculated with a suspension of freshly collected urediospores in distilled water containing Tween 20® (100 ml/L) approximately seven days after planting. Infection types (IT's) were scored 10 days post-inoculation (d.p.i.). Fully expanded flag leaves were inoculated on the upper surface. Due to differences in growth period adult plants were inoculated on 15 occasions. IT's and leaf rust severity were scored 16 d.p.i. Plant height, growth habit and head type of adult plants were also recorded. The number of days from planting to flag leaf stage varied from 54 to 187. One hundred and eighty two of the accessions were resistant to moderately resistant in the adult stage, whereas 126 were resistant or moderately resistant as seedlings to the pathotype mixture. IT's of accessions classified as resistant ranged between 0 and 2 (0 to 4 scale) and represented a wide range of phenotypes. High levels of resistance, typified by the absence of macroscopic symptoms, were observed in adult plants of *T. longissimum*, *T. sharonense*, *T. searsii* and *T. turgidum* ssp. *compactum*. *Triticum kotschyi* and *T. ventricosum* expressed hypersensitive IT's where small pustules were commonly associated with chlorosis and/or necrosis of leaf tissue. Partial resistance, expressed by small pustules without any apparent chlorosis, was observed in *T. turgidum* ssp. *durum*, *T. turgidum* ssp. *pyramidale* and *T. tauschii*. In *T. turgidum*, which comprised 14 subspecies and 272 accessions, approximately 44% of the adult plants were resistant to moderately resistant compared to 24% of the seedlings.

INTRODUCTION

The genus *Triticum* L. comprises diploid, tetraploid and hexaploid species. In earlier

years, several *Triticum* species were cultivated, however, production is now restricted almost entirely to tetraploid durum wheat (*T. turgidum* L.) and the hexaploid common or bread wheat (*T. aestivum* L.) (Knott, 1989a).

The ability of rust pathogens to mutate and form new and virulent races, necessitates the broadening of the genetic base of resistance in wheat to rust diseases. Virulence changes in leaf rust have been frequent (Samborski, 1982; Statler *et al.*, 1982; Pretorius, 1988) and the number of genes providing useful levels of resistance is limited (Browder, 1980; Kolmer, 1996). Furthermore, the low frequency of virulence to certain genes which condition resistance in wheat seedlings could only be ascribed to the limited use of those genes in commercial cultivars (Kolmer, 1996). In breeding programmes leaf rust resistance could be achieved through several approaches. Major gene resistance could be selected for in conventional or backcross populations, or alternatively, breeders could select against race-specificity and accumulate genes for slow rusting or adult-plant resistance in a cultivar. Irrespective of the approach followed, sources of resistance must be available for exploitation. The importance of species related to the cultivated *Triticum* species as gene sources for leaf rust resistance in wheat is well documented (Knott & Dvorak, 1976; Fraunstein & Hammer, 1985; Gill *et al.*, 1985; Valkoun *et al.*, 1985; Valkoun *et al.*, 1986; Manisterski *et al.*, 1988; Singh *et al.*, 1988; Kerber & Dyck, 1990; Damania *et al.*, 1991; Dhaliwal *et al.*, 1991; Dhaliwal *et al.*, 1993; Dimov *et al.*, 1993; Dyck and Bartoš, 1994; Antonov & Marais, 1996).

The aim of this study was to evaluate and characterise a collection of *Triticum* species for resistance to South African pathotypes of leaf rust caused by *P. recondita* Rob. ex Desm. f. sp. *tritici*.

MATERIALS AND METHODS

Accessions screened Four hundred and five *Triticum* accessions, comprising 13 diploid, tetraploid or hexaploid species, were obtained from the germplasm collection maintained by the Department of Genetics, University of Stellenbosch. Only 353 of these germinated and were tested for resistance to leaf rust. Species nomenclature is according to the Department of Genetics, University of Stellenbosch, whereas

authorities, where available, are according to Kimber & Sears (1987) and Van Slageren (1994).

Inoculum production Prior to inoculation, the South African pathotypes UVPrt 2, 3, 9 and 13 of *P. recondita* f. sp. *tritici* (Table 1) were produced on seedlings of susceptible cultivars/lines. Seedlings for rust multiplication were grown in 300 ml plastic pots containing a 1:1 v/v sterilised soil-peat moss mixture. To retard plant development and enhance sporulation, 50 ml of a 0.3 g/l maleic hydrazide solution were added per pot when seedlings emerged (Knott, 1989a). Seven days after planting seedlings were spray-inoculated with urediospores of each pathotype suspended (approximately 1 mg spores/ml) in light mineral oil (McSherry & Harris, Wedmore, Somerset, UK). Thereafter, plants were placed in the dark in a dew-simulation chamber for 16 h. Seedlings were then maintained in isolation cabinets on a glasshouse bench until urediospores were collected.

Seedling tests Approximately 10 seeds of each *Triticum* accession were grown as described above. Seedlings of each accession were inoculated with an equal mixture of freshly collected urediospores of pathotypes UVPrt 2, 3, 9 and 13 suspended (1mg/ml) in distilled water containing Tween 20®. Inoculated plants were incubated for 16 h in the dark in a dew chamber ($\pm$ 20°C). Thereafter plants were maintained in a growth chamber at 20°C. A 14 h daylength of 200 μmol m^{-2}s^{-1} photosynthetically active radiation was provided by fluorescent tubes and incandescent bulbs arranged 30 cm above plants. IT's were scored 10 d.p.i.

Adult-plant tests Three seeds of each accession were planted in a plastic pot containing 1L of soil and grown in leaf-rust-free, air-conditioned glasshouse cubicles at 25°C (day) and 15°C (night). Natural daylight was supplemented with 120 μEm^{-2}s^{-1} photosynthetically active radiation emitted by cool white fluorescent tubes, arranged directly above plants, for 14 h each day. Once plants reached the one and a half leaf stage, 50 ml per pot of a 3g/l hydroponic nutrient solution (6.5:2.7:13 N:P:K plus micro-elements), were added three times weekly as a soil drench for the duration of the experiment.

The upper surface of fully expanded flag leaves was spray-inoculated and plants incubated as described for the seedlings and maintained thereafter in a glasshouse at conditions similar to that prior to inoculation. Due to differences in growth period, sets of plants were inoculated on 15 occasions. IT's and leaf rust severity were scored 16 days post-inoculation.

Host reaction IT's were scored according to a 0 to 4 scale (Roelfs, 1988b) where 0=absence of macroscopic symptoms and 4=susceptibility. Flecks are indicated by ";" and chlorosis and necrosis by "C" and "N", respectively. Plus or minus signs indicate variation above or below established pustule sizes (McIntosh *et al.*, 1995). Severity was scored according to the modified Cobb scale and reaction classes were allocated according to Roelfs *et al.* (1992).

Genotype descriptions To distinguish among phenotypic appearance of accessions, head type was classified according to illustrations in Fig. 1 (Peterson, 1965; Miller, 1987). Plant height and days to full flag leaf expansion were also determined for each accession. Growth habit was classified as either erect, intermediate or prostrate.

RESULTS AND DISCUSSION

The expression of leaf rust resistance is given in Table 2. Most species/subspecies contained resistant accessions, either as seedlings, adult plants, or both. The exceptions were *T. cylindricum* (Host) Ces., Pass. & Gibelli, where IT's showed either an intermediate or susceptible reaction, and *T. turgidum* ssp. *carthlicum* (Nevski) Á. Löve & D. Löve (Table 3), which was susceptible to the leaf rust isolates used.

Of the 353 accessions tested as seedlings, 34 were resistant, 92 had intermediate IT's and 227 were susceptible (Table 3). High levels of resistance were observed in seedlings of *T. longissimum* (Schweinf. & Muschl.) Bowden, *T. turgidum* ssp. *dicoccum* Schrank *ex* Schübler var. rufum and *T. turgidum* ssp. *pyramidale* var. recognitum.

Resistance, typified by the absence of macroscopic symptoms, was observed in adult plants of *T. longissimum* (Fig. 2A), *T. sharonense* (Eig) Feldman & Sears, *T.*

searsii (Feldman & Kislev) Feldman and *T. turgidum* ssp. *compactum. Triticum kotschyi* (Boiss.) Bowden and *T. ventricosum* (Tausch) Ces., Pass. & Gilelli expressed hypersensitive reactions where small pustules were commonly associated with chlorosis and/or necrosis of leaf tissue. Partial resistance, expressed by smaller pustules without any
apparent chlorosis, was observed in species such as *T. turgidum* ssp. *durum* (Desf.) Husn., *T. turgidum* ssp. *pyramidale* and *T. tauschii* (Coss.) Schmal. (Fig. 2B). According to Lehman & Shaner (1996), this type of resistance to leaf rust may be more durable than high levels of hypersensitive resistance.

When seedling reactions were compared with those of flag leaves, adult-plant resistance (APR) was particularly noticeable in *T. tauschii, T. timopheevii* (Zhuk.) Zhuk. (Fig. 2C), *T. sharonense, T. turgidum* ssp. *anyleum* and *T. turgidum* ssp. *dicoccoides* (Körn. *ex* Aschers. & Graebner) Thell. One hundred and eighty two accessions were rated as resistant or moderately resistant to leaf rust
in the adult stage.

The expression of higher resistance levels in adult *T. longissimum* plants than in seedlings has previously been observed. In field tests conducted by Dhaliwal *et al.* (1993) at two locations, all *T. longissimum* accessions were resistant to leaf rust. Also, all adult *T. longissimum* plants tested by Manisterski *et al.* (1988) were resistant, whereas in seedling tests, approximately 47% of the accessions showed a resistant reaction. Similarly, Gill *et al.* (1985) and Antonov & Marais (1996) detected resistance in seedlings of some *T. longissimum* lines.

Results obtained by Manisterski *et al.* (1988) indicated that all adult plants of *T. sharonense* screened were resistant while only 45% of these showed a resistant seedling reaction. In seedling tests done by Gill *et al.* (1985) and Antonov & Marais (1996), only 25% and 31% of the *T. sharonense* accessions, respectively, were resistant to leaf rust. Studies on *T. searsii* showed 5% (Manisterski *et al.*, 1988), 66% (Gill *et al.,* 1985) and 38% (Antonov & Marais, 1996) of accessions tested resistant to wheat leaf rust. *Triticum kotschyi* showed a higher level of resistance in adult plants (Manisterski *et al.*, 1988; Dhaliwal *et al.*, 1993) than in seedlings (Gill *et al.*, 1985; Manisterski *et al.*, 1988; Antonov & Marais, 1996). No seedling resistance was expressed by accessions of *T. ventricosum* (Gill *et al.*, 1985; Antonov & Marais, 1996)

whereas in field tests conducted by Dhaliwal *et al.* (1993) 85.7 % showed APR. *Triticum turgidum* ssp. *dicoccoides* (Fig. 2D), which represented most accessions tested in the present study, resulted in 32% of the adult plants and 12% seedlings to be either resistant or moderately resistant.

Considerable variation in leaf rust resistance can be expected between and within species related to bread wheat and generalisations about the rust phenotypes, specifically between seedlings and mature plants, are difficult. However, based on the pathogenicity observed in the present study, as well as the reports mentioned above, it appears that resistance is more clearly detectable in adult plants. In other studies most of the related species tested for resistance to leaf rust were evaluated in the seedling stage (Moseman *et al.*, 1985; Gill *et al.*, 1986; Cox *et al.*, 1992; The *et al.*, 1993; Antonov & Marais, 1996; Brown-Guedira *et al.*, 1996; Brown-Guedira *et al.*, 1997) with the exception of Manisterski *et al.* (1988) who screened *Aegilops* and *Agropyron* species in both seedling and adult-plant stages and Dhaliwal *et al.* (1993) who screened several species in the field. Due to the emphasis on seedling resistance, it is thus possible that unidentified genes for APR may exsist in many related species. Some of the accessions tested (no's. 34, 46, 47, 56, 108, 164) (Table 2) showed a susceptible IT in the seedling stage and APR.

According to Manisterski *et al.* (1988) the lower levels of resistance observed on seedlings may be due to the higher spore concentrations usually applied in artificial inoculations. In the present study inoculum densities, normally used in the routine screening of wheat seedlings for rust resistance, were applied. Furthermore, severities of 70% and higher were often recorded on flag leaves during the primary uredinial phase, suggesting that high densities were similarly applied to adult plants. This implies that differentiation between seedling and adult-plant resistance, and between low and high levels of adult-plant resistance, were valid.

Results obtained by The *et al.* (1993) and Nevo (1993) indicated that *T. turgidum* ssp. *dicoccoides* generally does not have good seedling resistance to leaf rust. Dyck (1994) identified two accessions of *T. turgidum* ssp. *dicoccoides* with high levels of resistance against leaf rust and transferred two genes to the hexaploid wheat cultivar Thatcher. One of these genes was allelic to *Lr33* in line RL6057. The second gene may be a previously unidentified gene. Moseman *et al.* (1985) identified

potentially useful seedling resistance in *T. turgidum* ssp. *dicoccoides*. Also, the gene *Lr14a*, which can be detected in seedlings, was transferred from emmer to common wheat (McIntosh *et al.*, 1995). Some accessions of *T. turgidum* ssp. *dicoccoides*, e.g. no. 8 and 164, showed seedling susceptibility contrasted with low flag leaf IT's (0 and ;^{c} respectively). No adult-plant resistance genes have thus far been identified in *T. dicoccoides*, suggesting that some of the resistance observed may be due to genes not previously described.

Triticum cylindricum accessions expressed either an intermediate or susceptible reaction to leaf rust at both growth stages. Dhaliwal *et al.* (1993) tested *T. cylindricum* for resistance to leaf rust under natural field conditions and recorded resistance in 41.4% and 36.6% of the accessions, depending on the location. Seedling tests conducted by Antonov & Marais (1996) indicated that only 13% *T. cylindricum* accessions were resistant when tested with a mixture of pathotypes UVPrt2, 3, 8, 9 and 13. Dimov *et al.* (1993) found no resistance in 27 accessions of *T. cylindricum* tested in both the seedling and adult stages. However, Bai *et al.* (1995) found that seedlings of three accessions of *T. cylindricum* were resistant to ten races of leaf rust while Gill *et al.* (1985) indicated 91% resistance in seedlings of the same species.

In *T. timopheevii* only one accession was resistant in the seedling stage, whilst 13 accessions were resistant as adult plants. Resistance to leaf rust has previously been found in *T. timopheevii* (Gill *et al.*, 1983; Dhaliwal *et al.*, 1993; Antonov & Marais, 1996; Brown-Guedira *et al.*, 1996; Brown-Guedira *et al.*, 1997). Only one catalogued gene (*Lr18*) has been transferred to common wheat from this species (McIntosh *et al.*, 1995). *Triticum timopheevii* crosses readily with bread wheat, although the degree of difficulty varies with different crosses. According to Knott (1989a), the best seed set is obtained when *T. timopheevii* is used as the male parent. Viable hybrid seed can be recovered from crosses between hexaploid wheat and *T. araraticum*, which is taxonomically related to *T. timopheevii*, but male sterility and even complete sterility is often a problem in backcrosses (Brown-Guedira *et al.*, 1997). Considering that *T. timopheevii* has been an important source of cytoplasmic male sterility for hybrid breeding and also for fertility-restoring genes, the occurrence of male sterility is not surprising (Knott, 1989a). *Triticum timopheevii* carries an A genome plus a distinct genome designated G. Genes in the A genome should be easier to transfer than those

situated on the G genome (Knott, 1987).

In *T. tauschii*, one of the wild ancestors of common wheat, 88% of accessions tested as adult plants and seedlings were resistant to wheat leaf rust. This species has been a rich reservoir of wheat leaf rust resistance and several known *Lr* genes have been transferred from *T. tauschii* to common wheat, namely *Lr21*, *Lr22a*, *Lr32*, *Lr39*, *Lr40*, *Lr41*, *Lr42* and *Lr43* (Kerber & Dyck, 1969; Dyck & Kerber, 1970; Rowland & Kerber, 1974; Kerber, 1987; Cox *et al.*, 1992a; Cox *et al.*, 1994; McIntosh *et al.*, 1995).

To provide more information to breeders wishing to utilise these accessions as leaf rust resistant donors, full descriptions according to 16 different head types (Fig. 1), growth habit, the number of days from planting to flag leaf stage and plant height are provided in Table 2. *Triticum tauschii*, *T. bicorne* Forssk., *T. cylindricum, T. juvenale* Thell., *T. kotschyi, T. longissimum, T. syriacum* Bowden and *T. peregrinum* Hackel in J. Fraser had prostrate growth habits. However, some accessions of *T. tauschii* had intermediate growth habits and some accessions of *T. longissimum* were either intermediate or erect. Accessions of *T. timopheevii, T. ventricosum, T. sharonense* and *T. searsii* had an intermediate growth habit, although some *T. sharonense* lines had an erect appearance. Almost all accessions of *T. dicoccoides* had intermediate growth habits. Most *T. turgidum* subspecies had erect growth habits, except the subspecies *anyleum, durum, compactum, dicoccum, orientale* and *polonicum* (L.) Thell. The number of days from planting to flag leaf stage varied from 54 to 187 (Table 2).

This study indicated that wild relatives have considerable potential as sources of leaf rust resistance. It is possible that resistance identified in this study may be allelic to genes already transferred to bread wheat, but the possibility of previously undescribed genes should be investigated further. Keeping in mind the difficulty of transfer, it is possible that the choice of the donor species for improvement of cultivated wheats may be restricted to closely related species even if higher levels of resistance exist in more distant ones.

Table 1. Avirulence/virulence[a] combinations of South African pathotypes of *Puccinia recondita* f. sp. *tritici* used for inoculation

Pathotype	Leaf rust resistance (*Lr*) genes[b]	Selective hosts[c]
UVPrt2	*Lr1,2a,2b,3ka,11,15,17,20,24,26,30/2c,3a,3bg,10,14a,16*	Zaragosa
UVPrt3	*Lr3a,3bg,3ka,10,11,14a,16,17,20,26,30/1,2a,2b,2c,15,24*	Agent
UVPrt9	*Lr2a,2b,3bg,15,16,17,26,30/1,2a,2b,2c,10,14a,15,17,24*	Karee
UVPrt13	*Lr3a,3bg,3ka,11,16,20,30/1,2a,2b,2c,10,14a,15,17,24,26*	RL6078

[a] Avirulence/virulence characteristics determined at 18-24°C.
[b] South African leaf rust differentiating genes.
[c] Selective lines on which pathotypes were increased.

Table 2. Evaluation of a collection of wild *Triticum* species for resistance to *Puccinia recondita* f. sp. *tritici*[a] and criteria used to differentiate among accessions

Accession Number	Species[b]	Seedling infection type[c]	Adult plant infection type[c]	Adult plant leaf rust severity and reaction type[d]	Days to full flag leaf[e]	Plant height (cm)	Head type[f]	Growth habit[g]
1	*T. tauschii* var. typica squarossa	3[++]	3[+]	80S	69	50	14	A
2	*T. tauschii* var. strangulata squarossa	X	;	0R	160	76	14	A
3	*T. tauschii* var. strangulata squarossa	2	;[c]	5R	160	57	14	A
4	*T. tauschii*	3	2	60MS	77	49	14	A
49	*T. tauschii*	2	2	necrotic	77	31	---[h]	A
50	*T. tauschii*	2	1[++]	80MS-S	75	64	14	A
76	*T. tauschii*	2	;	tR	172	50	14	B
309	*T. tauchii*	2[+]	3	60MS-S	111	36	14	B
310	*T. tauschii*	2[+]	1	40MR-MS	132	62	14	B
311	*T. tauschii*	2[+]	3[-]	40MS-S	111	45	14	B
313	*T. tauschii*	2[-]	4	80S	75	114	14	B
316	*T. tauschii*	2	;1[-]	5R-MR	153	62	14	B
329	*T. tauschii*	2	;	0R	187	60	14	A
344	*T. tauschii*	2[-]	; ;1[-]	20MR	172	57	14	A
345	*T. tauschii*	2	;	0R	187	51	14	A
389	*T. tauschii*	23	;	tR	172	61	14	A
390	*T. tauschii*	2	;1[-]	10MR	172	67	14	A
30	*T. timopheevii* ssp. *araraticum* var. tumanianii	3[++]	3	40MS	187	105	9	B
33	*T. timopheevii* ssp. *araraticum*	2	;1	10MR	160	84	9	B
34	*T. timopheevii* ssp. *araraticum*	3	;[c]	5R	153	78	9	B

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Table 2 (cont.). Evaluation of a collection of wild *Triticum* species for resistance to *Puccinia recondita* f. sp. *tritici*[a] and criteria used to differentiate among accessions

Accession Number	Species[b]	Seedling infection type[c]	Adult plant infection type[c]	Adult plant leaf rust severity and reaction type[d]	Days to full flag leaf[e]	Plant height (cm)	Head type[f]	Growth habit[g]
77	*T. timopheevii* ssp. *araraticum* var. nachitchevanicum	3	4	80S	104	93	8	B
79	*T. timopheevii* ssp. *araraticum* var. nachitchevanicum	2	;	tR	125	92	8	B
80	*T. timopheevii* ssp. *araraticum* var. nachitchevanicum	2	;1	10MR	118	95	8	B
81	*T. timopheevii* ssp. *araraticum* var. nachitchevanicum	3	3⁻	10MS	118	102	8	B
82	*T. timopheevii* ssp. *araraticum* var. nachitchevanicum	3⁺	;1⁻	tR	125	103	8	B
83	*T. timopheevii* ssp. araraticum var. nachitchevanicum	2	;	5R	118	93	8	B
84	*T. timopheevii* ssp. *araraticum* var. nachitchevanicum	3⁻	3	80MS-S	132	101	8	B
85	*T. timopheevii* ssp. *araraticum* var. nachitchevanicum	3	1⁺⁺	40MS	153	89	8	B
86	*T. timopheevii* ssp. *araraticum* var. nachitchevanicum	3	2⁺	40MS	132	104	8	B
87	*T. timopheevii* ssp. *araraticum* var. tumanianii	3	1⁺⁺	20MR-MS	125	94	8	B
88	*T. timopheevii* ssp. *araraticum* var. tumanianii	3	3⁻	40MS-S	153	102	8	B
89	*T. timopheevii* ssp. *araraticum* var. tumanianii	2	;1⁺	40MR	153	112	8	B
305	*T. timopheevii* ssp. *araraticum*	3⁺	;1	20MR	132	97	9	B
324	*T. timopheevii* var. typicum	23	2	80MS	75	142	9	B
362	*T. timopheevii*	;-3	;1⁺	10MR	54	98	9	C
35	*T. bicorne*	3	4	50S	89	56	6	A
36	*T. bicorne*	3	3	20S	104	41	6	A
37	*T. bicorne*	3	4	80S	89	38	6	A
53	*T. bicorne*	3⁺	;1	20MR	111	51	6	A
54	*T. bicorne*	3	4	80S	89	51	6	A

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Table 2 (cont.). Evaluation of a collection of wild *Triticum* species for resistance to *Puccinia recondita* f. sp. *tritici*[a] and criteria used to differentiate among accessions

Accession Number	Species[b]	Seedling infection type[c]	Adult plant infection type[c]	Adult plant leaf rust severity and reaction type[d]	Days to full flag leaf[e]	Plant height (cm)	Head type[f]	Growth habit[e]
55	*T. bicorne*	3	4	80S	89	54	6	A
326	*T. bicorne* var. typicum	3	3	20S	104	42	6	A
331	*T. bicorne*	3	;1	20MR	132	50	6	B
384	*T. bicorne*	2	2[c--]	40MR	125	47	6	B
385	*T. bicorne*	3[+]	3[+]	50S	111	57	6	B
39	*T. cylindricum*	3	2	80MS-S	75	43	13	A
58	*T. cylindricum*	2	3	80S	89	46	13	A
40	*T. juvenale*	3	4	80S	89	36	13	A
41	*T. juvenale*	3[+]	3[c]	50S	54	35	13	A
335	*T. juvenale*	3[+]	3[+]	60S	125	97	15	B
343	*T. juvenale*	2[-]	;1[cn]	40R-MR	89	55	8	A
42	*T. kotcshyi*	2	;1[cn]	20MR	75	26	16	A
43	*T. kotcshyi*	2	;1[cn]	20MR	75	38	16	A
334	*T. kotschyi*	;	;	tR	153	61	16	A
44	*T. longissimum*	1[c]	0	0R	77	48	15	A
45	*T. longissimum*	2	;	tR	111	124	15	C
46	*T. longissimum*	3	;	tR	89	50	15	A
51	*T. longissimum*	;1	;1	5MR	75	127	15	B
52	*T. longissimum*	;1	;	5R	69	79	15	A
304	*T. longissimum*	;1	;	tR	125	94	15	C

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Table 2 (cont.). Evaluation of a collection of wild *Triticum* species for resistance to *Puccinia recondita* f. sp. *tritici*[a] and criteria used to differentiate among accessions

Accession Number	Species[b]	Seedling infection type[c]	Adult plant infection type[c]	Adult plant leaf rust severity and reaction type[d]	Days to full flag leaf[e]	Plant height (cm)	Head type[f]	Growth habit[g]
336	*T. longissimum*	;1	;^{cn}	5R	153	121	15	B
47	*T. sharonense*	3^{+c}	;	tR	125	98	6	C
48	*T. sharonense*	2,3	;	10R	104	116	6	C
56	*T. sharonense*	3$^+$	;	5R	111	84	6	B
327	*T. sharonense* var. typica	;1	;	tR	125	98	15	C
342	*T. sharonense*	;-3	; - ;1	tR	132	114	6	B
375	*T. sharonense*	23	;	5R	118	106	6	B
395	*T. sharonense*	;	;	tR	118	87	12	B
73	*T. searsii*	;1	;	tR	104	67	15	C
74	*T. searsii*	;1	;	tR	132	57	15	B
374	*T. searsii*	23	;1	5MR	153	61	15	B
382	*T. searsii*	2	; - ;1	5MR	132	62	15	B
383	*T. searsii*	3	;1$^+$	10MR	153	42	15	B
303	*T. ventricosum*	2	;1^c	10R-MR	153	52	13	B
332	*T. ventricosum*	2$^-$	;1^{-c}	10R-MR	153	47	13	B
386	*T. syriacum*	2	2^c	70MS	89	74	13	A
387	*T. syriacum*	2	2^c	70MS	89	50	13	A
388	*T. syriacum*	2	;1	20R-MR	104	70	13	A
333	*T. peregrinum*	;1	;1^{cn}	20MR	75	40	16	A
142	*T. turgidum* var. nigro-barbatum	3	;1$^+$	20MR	75	143	2	C

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Table 2 (cont.). Evaluation of a collection of wild *Triticum* species for resistance to *Puccinia recondita* f. sp. *tritici*[a] and criteria used to differentiate among accessions

Accession Number	Species[b]	Seedling infection type[c]	Adult plant infection type[c]	Adult plant leaf rust severity and reaction type[d]	Days to full flag leaf[e]	Plant height (cm)	Head type[f]	Growth habit[g]
102	*T. turgidum* ssp. *dicoccum* var. arras	2⁻	3ᶜ	70MS-S	54	84	10	C
103	*T. turgidum* ssp. *dicoccum* var. farrum	2⁻	3	70MS-S	54	87	10	C
104	*T. turgidum* ssp. *dicoccum* var. arras	x	;	tR	89	140	9	B
105	*T. turgidum* ssp. *dicoccum* var. farrum	;1	;	tR	89	130	10	B
347	*T. turgidum* ssp. *dicoccum*	;-3	;1ᶜ	60R-MR	63	97	1	C
348	*T. turgidum* ssp. *dicoccum*	;-3	;1ᶜ	60R-MR	63	116	10	C
354	*T. turgidum* ssp. *dicoccum* var. rufum	;1	;1	20MR	54	118	1	C
355	*T. turgidum* ssp. *dicoccum* var. rufum	;1	;ᶜ	20R	75	110	1	C
356	*T. turgidum* ssp. *dicoccum* var. rufum	;1*	2	40MS	75	112	1	C
367	*T. turgidum* ssp. *dicoccocum* var. khapli	;1**	4	60S	54	52	10	C
108	*T. turgidum* ssp. *anyleum* var. atratum	3**	;ᶜⁿ	5R	172	105	7	B
109	*T. turgidum* ssp. *anyleum* var. rufum	2*	;ᶜ	50R	69	147	8	B
110	*T. turgidum* ssp. *anyleum* var. rufum	;1*	2	40MS	75	127	7	B
111	*T. turgidum* ssp. *anyleum* var. rufum	2*	;1	20MR	77	110	8	B
112	*T. turgidum* ssp. *anyleum* var. tricoccum	3	2	20MS	77	126	8	B
113	*T. turgidum* ssp. *anyleum* var. nigro-ajar	;1ᶜ	;ᶜ	30R	69	124	7	B
114	*T. turgidum* ssp. *anyleum* var. macroanterum	3*	1**	40MR-MS	153	103	3	B
115	*T. turgidum* ssp. *anyleum*	2	;1	5MR	77	113	7	B
116	*T. turgidum* ssp. *abyssinicum*	;1*	;	20R	77	158	2	B
117	*T. turgidum* ssp. *palaeocolchicum* var. schwamilicum	2	;ᶜ	5R	172	105	11	C

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Table 2 (cont.). Evaluation of a collection of wild *Triticum* species for resistance to *Puccinia recondita* f. sp. *tritici*[a] and criteria used to differentiate among accessions

Accession Number	Species[b]	Seedling infection type[c]	Adult plant infection type[c]	Adult plant leaf rust severity and reaction type[d]	Days to full flag leaf[e]	Plant height (cm)	Head type[f]	Growth habit[g]
118	*T. turgidum* ssp. *palaeocolchicum* var. schwamilicum	2	;1[+]	30MR	160	115	11	C
119	*T. turgidum* ssp. *palaeocolchicum*	2[++]	;[c]	5R	172	114	11	C
120	*T. turgidum* ssp. *palaeocolchicum*	2	;1[+]	30MR	160	118	11	C
337	*T. turgidum* ssp. *palaeocolchicum*	;12,3	;1	20MR	75	104	9	B
121	*T. turgidum* ssp. *durum* var. reichenbachii	;1[−]	;	tR	75	134	1	B
122	*T. turgidum* ssp. *durum* var. hordeiforme [a]	;1	;1	20MR	69	129	1	B
123	*T. turgidum* ssp. *durum* var. melanopus	2[+]	1[++]	20MS	77	131	1	B
124	*T. turgidum* ssp. *durum* var. africanum	3[+]	3	70S	54	139	1	B
125	*T. turgidum* ssp. *durum* var. murciense	2	;1	5R-MR	69	117	1	B
126	*T. turgidum* ssp. *durum* var. obscurum	2,3	1	5MR	77	108	1	B
127	*T. turgidum* ssp. *durum* var. aestivum	1	;	5R	69	110	1	B
128	*T. turgidum* ssp. *durum* var. duro-compactum	1[+]	4	80S	77	106	1	B
129	*T. turgidum* ssp. *durum* var. libycum	2[+],3	;1	20MR	75	108	1	B
130	*T. turgidum* ssp. *persicum* var. stramineum	3[++]	2[c]	40MR-MS	77	134	10	C
131	*T. turgidum* ssp. *persicum* var. stramineum	3[−]	;1[c]	20MR	77	120	10	C
132	*T. turgidum* ssp. *persicum* var. stramineum	3[++]	1[++]	20MS	77	110	10	C
133	*T. turgidum* ssp. *persicum* var. fuliginosum	3	2[−]	40MR-MS	75	111	10	C
134	*T. turgidum* ssp. *persicum* var. rubiginosum	3	2[c]	70MS	77	90	10	C
135	*T. turgidum* ssp. *persicum*	3	3[−]	80MS-S	77	115	10	C
136	*T. turgidum* ssp. *persicum*	2	2[−]	60MR-MS	77	130	10	C

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Table 2 (cont.). Evaluation of a collection of wild *Triticum* species for resistance to *Puccinia recondita* f. sp. *tritici*[a] and criteria used to differentiate among accessions

Accession Number	Species[b]	Seedling infection type[c]	Adult plant infection type[c]	Adult plant leaf rust severity and reaction type[d]	Days to full flag leaf[e]	Plant height (cm)	Head type[f]	Growth habit[g]
137	*T. turgidum* ssp. *persicum*	3	2⁻	50MS	75	119	10	C
349	*T. turgidum* ssp. *persicum*	3	4	80S	63	106	10	C
350	*T. turgidum* ssp. *persicum*	;13	3⁻	60MS-S	75	78	10	C
351	*T. turgidum* ssp. *persicum* var. *stramineum*	;13	;1*	40MR-MS	63	116	10	C
352	*T. turgidum* ssp. *persicum* var. *stramineum*	3	3	80S	54	103	10	C
353	*T. turgidum* ssp. *persicum* var. *rubiginosum*	3⁺	;1	30MR	63	118	10	C
400	*T. turgidum* ssp. *persicum*	2	2*	50MS	77	105	10	C
401	*T. turgidum* ssp. *persicum*	2	2	50MR-MS	75	110	10	C
138	*T. turgidum* ssp. *pyramidale* var. *recognitum*	X⁻	1	10MR	54	104	11	C
139	*T. turgidum* ssp. *pyramidale* var. *recognitum*	X⁻	1	10MR	54	90	11	C
140	*T. turgidum* ssp. *pyramidale* var. *compacticum*	3⁻	3	50MS-S	63	88	11	C
141	*T. turgidum* ssp. *pyramidale*	;-2	1	5MR	54	87	11	C
318	*T. turgidum* ssp. *pyramidale*	;1⁻	;	tR	75	116	10	C
143	*T. turgidum* ssp. *compactum*	1	;	0R	69	146	3	B
144	*T. turgidum* ssp. *compactum*	1	;	tR	75	131	3	B
145	*T. turgidum* ssp. *orientale* var. *insigne*	4	3**	80S	69	102	5	C
146	*T. turgidum* ssp. *orientale* var. *notabile*	3	;1*	20MR	118	114	5	C
321	*T. turgidum* ssp. *orientale* var. *insigne*	3⁻	4	80S	75	106	5	B
322	*T. turgidum* ssp. *orientale* var. *notabile*	3⁻	4	80S	77	112	5	B
371	*T. turgidum* ssp. *orientale* var. *euisgne*	23	;1	20MR	132	106	10	C

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Table 2 (cont.). Evaluation of a collection of wild *Triticum* species for resistance to *Puccinia recondita* f. sp. *tritici*[a] and criteria used to differentiate among accessions

Accession Number	Species[b]	Seedling Infection type[c]	Adult plant infection type[c]	Adult plant leaf rust severity and reaction type[d]	Days to full flag leaf[e]	Plant height (cm)	Head type[f]	Growth habit[g]
147	*T. turgidum* ssp. *aethiopicum*	;1[c]	;1[cn]	40MR	54	74	4	C
148	*T. turgidum* ssp. *aethiopicum*	2[+],3	3[c]	60MS-S	54	101	4	C
325	*T. turgidum* ssp. *aethiopicum* var. atratosanquineum	3	2[c]	50MS	54	80	10	C
149	*T. turgidum* ssp. *polonicum* var. vesticum	3[++]	;1	20MR	75	116	5	B
323	*T. turgidum* ssp. *polonicum* var. levissimum	2,3	2[*]	60MS-S	89	116	5	B
338	*T. turgidum* ssp. *polonicum*	23	;1[c]	15MR	54	125	5	B
363	*T. turgidum* ssp. *polonicum*	3[−]	3	80MS	54	115	5	B
365	*T. turgidum* ssp. *polonicum*	23	3[-]	80S	69	116	5	B
368	*T. turgidum* ssp. *polonicum* var. vestitum	23	;1	5MR	77	121	5	B
370	*T. turgidum* ssp. *polonicum*	2[−]	;	10R	104	118	5	B
150	*T. turgidum* ssp. *isphahanicum*	z	;	tR	54	111	5	B
339	*T. turgidum* ssp. *carthlicum*	3	4	80S	54	125	5	B
369	*T. turgidum* ssp. *carthlicum* var. stramineum	23	2[*]	60MS-S	75	98	10	C
6	*T. turgidum* ssp. *dicoccoides*	4	3[++]	80S	153	104	8	B
8	*T. turgidum* ssp. *dicoccoides*	3[+]	0	0R	125	94	8	B
9	*T. turgidum* ssp. *dicoccoides*	3	4	80S	118	65	8	B
11	*T. turgidum* ssp. *dicoccoides*	3	3[-c]	30MS	104	86	8	B
12	*T. turgidum* ssp. *dicoccoides*	3	4	80S	104	88	8	B
13	*T. turgidum* ssp. *dicoccoides*	3[+]	4	80S	75	120	8	B
14	*T. turgidum* ssp. *dicoccoides*	3	3	60S	104	71	8	B

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Table 2 (cont.). Evaluation of a collection of wild *Triticum* species for resistance to *Puccinia recondita* f. sp. *tritici*[a] and criteria used to differentiate among accessions

Accession Number	Species[b]	Seedling infection type[c]	Adult plant infection type[c]	Adult plant leaf rust severity and reaction type[d]	Days to full flag leaf[e]	Plant height (cm)	Head type[f]	Growth habit[g]
16	*T. turgidum* ssp. *dicoccoides*	3[++]	4	80S	89	108	8	B
17	*T. turgidum* ssp. *dicoccoides*	3[++]	3[c]	60MS	104	69	8	B
18	*T. turgidum* ssp. *dicoccoides*	3[+]	3[c]	60MS-S	104	90	8	B
19	*T. turgidum* ssp. *dicoccoides*	3[c]	2[c]	20MR-MS	125	82	8	B
20	*T. turgidum* ssp. *dicoccoides*	3[+]	4	80S	89	86	8	B
21	*T. turgidum* ssp. *dicoccoides*	2[+]	;1	necrotic	118	78	8	B
22	*T. turgidum* ssp. *dicoccoides*	3[++]	2;[+]	50MS	104	98	8	B
25	*T. turgidum* ssp. *dicoccoides*	3	3[+]	50S	104	93	8	B
26	*T. turgidum* ssp. *dicoccoides*	3[++]	4	80S	125	96	8	B
29	*T. turgidum* ssp. *dicoccoides*	3[++]	2[+]	40MS	160	106	8	B
60	*T. turgidum* ssp. *dicoccoides* var. *kotchianum*	3[++]	3[+c]	60MS-S	104	83	8	B
61	*T. turgidum* ssp. *dicoccoides* var. *spontaneonigrum*	3[+]	3[c]	70MS-S	104	94	8	B
62	*T. turgidum* ssp. *dicoccoides* var. *straussianum*	3[++]	1[++]	30MS	118	83	8	B
63	*T. turgidum* ssp. *dicoccoides* var. *arabicum*	3	4	80S	69	125	8	B
64	*T. turgidum* ssp. *dicoccoides* var. *spontaneovillosum*	3[++]	4	80S	69	108	8	B
65	*T. turgidum* ssp. *dicoccoides* var. *kotchianum*	3	3[+]	50S	187	96	8	B
70	*T. turgidum* ssp. *dicoccoides*	3[-]	2[c]	70MS	77	86	8	C
71	*T. turgidum* ssp. *dicoccoides*	3[+]	3	50S	69	135	8	B
72	*T. turgidum* ssp. *dicoccoides*	2	;1[c]	20MR	77	136	10	C
75	*T. turgidum* ssp. *dicoccoides*	3[++]	4	80S	77	102	8	B

Table 2 (cont.). Evaluation of a collection of wild *Triticum* species for resistance to *Puccinia recondita* f. sp. *tritici*[a] and criteria used to differentiate among accessions

Accession Number	Species[b]	Seedling infection type[c]	Adult plant infection type[c]	Adult plant leaf rust severity and reaction type[d]	Days to full flag leaf[e]	Plant height (cm)	Head type[f]	Growth habit[g]
90	*T. turgidum* ssp. *dicoccoides* var. kotchianum	3	3	50MS-S	172	120	8	B
91	*T. turgidum* ssp. *dicoccoides* var. kotchianum	3*	;1	20MR	69	116	8	B
93	*T. turgidum* ssp. *dicoccoides* var. spontaneovillosum	3	2++c	40MS	187	91	8	B
94	*T. turgidum* ssp. *dicoccoides* var. aaronsohni	3*	3*	50S	160	102	8	B
95	*T. turgidum* ssp. *dicoccoides* var. aaronsohni	3*	3++	70S	153	112	8	B
96	*T. turgidum* ssp. *dicoccoides* var. fulvo-villosum	3*	3*	70S	160	98	8	B
97	*T. turgidum* ssp. *dicoccoides* var. fulvo-villosum	3++	4	80S	153	96	8	B
98	*T. turgidum* ssp. *dicoccoides* var. spontaneonigrum	2	3-	50MS-S	75	102	8	B
99	*T. turgidum* ssp. *dicoccoides* var. aaronsohni	3++	4	80S	89	85	8	B
100	*T. turgidum* ssp. *dicoccoides* var. fulvo-villosum	2*	2c	70S	69	118	8	B
101	*T. turgidum* ssp. *dicoccoides* var. kotchianum	3	3*	70S	160	98	10	B
151	*T. turgidum* ssp. *dicoccoides*	3*	1**	40MS	125	78	8	B
152	*T. turgidum* ssp. *dicoccoides*	3*	3	30S	153	102	8	B
153	*T. turgidum* ssp. *dicoccoides*	3**	3	40S	153	79	8	B
154	*T. turgidum* ssp. *dicoccoides*	3**	3	40S	153	78	8	B
155	*T. turgidum* ssp. *dicoccoides*	3**	2	30MS	153	96	8	B
156	*T. turgidum* ssp. *dicoccoides*	3	3-	60MS-S	125	66	8	B
157	*T. turgidum* ssp. *dicoccoides*	3**	3-	60MS-S	125	74	8	B
158	*T. turgidum* ssp. *dicoccoides*	3**	3	40MS-S	153	76	8	B
159	*T. turgidum* ssp. *dicoccoides*	3**	3*	40S	153	81	8	B

Table 2 (cont.). Evaluation of a collection of wild *Triticum* species for resistance to *Puccinia recondita* f. sp. *tritici*[a] and criteria used to differentiate among accessions

Accession Number	Species[b]	Seedling infection type[c]	Adult plant Infection type[c]	Adult plant leaf rust severity and reaction type[d]	Days to full flag leaf[e]	Plant height (cm)	Head type[f]	Growth habit[g]
160	*T. turgidum* ssp. *dicoccoides*	3[++]	2	30MS	153	85	8	B
161	*T. turgidum* ssp. *dicoccoides*	3[+]	3	40S	153	76	8	B
162	*T. turgidum* ssp. *dicoccoides*	3[+]	3[+]	50S	153	68	8	B
163	*T. turgidum* ssp. *dicoccoides*	3[+]	3	50MS-S	125	71	8	B
164	*T. turgidum* ssp. *dicoccoides*	3[+]	;[c]	5R	172	88	8	B
165	*T. turgidum* ssp. *dicoccoides*	3[++]	3[++c]	60MS-S	187	93	8	B
166	*T. turgidum* ssp. *dicoccoides*	3[++]	;1[cn]	5R-MR	187	92	8	B
167	*T. turgidum* ssp. *dicoccoides*	3[++]	3	40S	160	86	8	B
168	*T. turgidum* ssp. *dicoccoides*	3[++]	3[-c]	60MS	160	98	8	B
169	*T. turgidum* ssp. *dicoccoides*	3[+]	4	80S	63	106	10	B
170	*T. turgidum* ssp. *dicoccoides*	2[+]	;1	20MR	172	102	8	B
172	*T. turgidum* ssp. *dicoccoides*	3	2[++c]	50MS	187	74	8	B
173	*T. turgidum* ssp. *dicoccoides*	2	3	40MS-S	153	98	8	B
174	*T. turgidum* ssp. *dicoccoides*	2[+],3	3[+]	60S	132	96	8	B
175	*T. turgidum* ssp. *dicoccoides*	2[+],3	3[-]	40MS	160	89	8	B
176	*T. turgidum* ssp. *dicoccoides*	2[+],3	1	10MR	125	96	8	B
177	*T. turgidum* ssp. *dicoccoides*	2[c]	2[+cn]	60MS	54	89	10	B
178	*T. turgidum* ssp. *dicoccoides*	3	3	80MS-S	104	110	10	B
179	*T. turgidum* ssp. *dicoccoides*	2[+],3	;1[-c]	10MR	153	112	8	B
180	*T. turgidum* ssp. *dicoccoides*	3	2[c]	30MS	153	137	8	B

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Table 2 (cont.). Evaluation of a collection of wild *Triticum* species for resistance to *Puccinia recondita* f. sp. *tritici*[a] and criteria used to differentiate among accessions

Accession Number	Species[b]	Seedling infection type[c]	Adult plant infection type[c]	Adult plant leaf rust severity and reaction type[d]	Days to full flag leaf[e]	Plant height (cm)	Head type[f]	Growth habit[g]
181	*T. turgidum* ssp. *dicoccoides*	3[*]	3[**]	70S	153	107	8	B
182	*T. turgidum* ssp. *dicoccoides*	3[*]	;1	20MR	153	102	8	B
183	*T. turgidum* ssp. *dicoccoides*	3[*]	2[*]	50MS	132	126	8	B
184	*T. turgidum* ssp. *dicoccoides*	3[*]	2	50MS	132	103	8	B
185	*T. turgidum* ssp. *dicoccoides*	3	2[c]	40MS	132	112	8	B
186	*T. turgidum* ssp. *dicoccoides*	3[*]	3[-]	40MS-S	118	109	8	B
187	*T. turgidum* ssp. *dicoccoides*	3[*]	3[*]	90S	118	87	8	B
188	*T. turgidum* ssp. *dicoccoides*	3[*]	3	40MS	132	116	8	B
190	*T. turgidum* ssp. *dicoccoides*	3[*]	2	40MS	132	107	8	B
191	*T. turgidum* ssp. *dicoccoides*	3[**]	2[**]	20MR-MS	153	111	8	B
192	*T. turgidum* ssp. *dicoccoides*	3[*]	3[-]	50MS-S	104	106	8	B
193	*T. turgidum* ssp. *dicoccoides*	3	1[**]	30MS	118	104	8	B
194	*T. turgidum* ssp. *dicoccoides*	3	1[c]	30MR	132	123	8	B
195	*T. turgidum* ssp. *dicoccoides*	3	2[*]	30MS	118	83	8	B
196	*T. turgidum* ssp. *dicoccoides*	3	2	30MS	132	118	8	B
197	*T. turgidum* ssp. *dicoccoides*	2[*]	1[*]	20MR	125	97	8	B
198	*T. turgidum* ssp. *dicoccoides*	3[*]	4	80S	69	116	8	B
199	*T. turgidum* ssp. *dicoccoides*	3	4	80S	63	97	8	B
200	*T. turgidum* ssp. *dicoccoides*	3	2	50MR-MS	104	93	8	B
201	*T. turgidum* ssp. *dicoccoides*	3[**]	4	80S	63	103	8	B

Table 2 (cont.). Evaluation of a collection of wild *Triticum* species for resistance to *Puccinia recondita* f. sp. *tritici*[a] and criteria used to differentiate among accessions

Accession Number	Species[b]	Seedling infection type[c]	Adult plant Infection type[c]	Adult plant leaf rust severity and reaction type[d]	Days to full flag leaf[e]	Plant height (cm)	Head type[f]	Growth habit[g]
202	*T. turgidum* ssp. *dicoccoides*	3**	4	80S	75	83	8	B
203	*T. turgidum* ssp. *dicoccoides*	3	2⁻	40MR	104	109	8	B
204	*T. turgidum* ssp. *dicoccoides*	3	3	60MR-MS	104	104	8	B
205	*T. turgidum* ssp. *dicoccoides*	3	x	40MS-S	111	94	8	B
206	*T. turgidum* ssp. *dicoccoides*	3*	4	80S	69	105	8	B
207	*T. turgidum* ssp. *dicoccoides*	3*	4	80S	69	101	8	B
208	*T. turgidum* ssp. *dicoccoides*	3*	4	80S	63	115	8	B
209	*T. turgidum* ssp. *dicoccoides*	3*	4	80S	63	99	8	B
210	*T. turgidum* ssp. *dicoccoides*	3*	4	80S	63	87	8	B
211	*T. turgidum* ssp. *dicoccoides*	3*	4	80S	75	85	8	B
212	*T. turgidum* ssp. *dicoccoides*	3*	4	80S	69	102	8	B
213	*T. turgidum* ssp. *dicoccoides*	3*	3	60MS-S	104	103	8	B
214	*T. turgidum* ssp. *dicoccoides*	3**	4	80S	63	91	8	B
215	*T. turgidum* ssp. *dicoccoides*	3	2**	70MS	187	101	8	B
216	*T. turgidum* ssp. *dicoccoides*	3**	4	80S	111	67	8	B
217	*T. turgidum* ssp. *dicoccoides*	3	2**c	50MS	187	86	8	B
218	*T. turgidum* ssp. *dicoccoides*	3	1**	20MR	132	69	8	B
219	*T. turgidum* ssp. *dicoccoides*	3**	3*c	50MS	160	96	8	B
220	*T. turgidum* ssp. *dicoccoides*	3	2*	40MS	172	116	8	B
222	*T. turgidum* ssp. *dicoccoides*	3*	2*	40MS	172	103	8	B

Table 2 (cont.). Evaluation of a collection of wild *Triticum* species for resistance to *Puccinia recondita* f. sp. *tritici*[a] and criteria used to differentiate among accessions

Accession Number	Species[b]	Seedling infection type[c]	Adult plant infection type[c]	Adult plant leaf rust severity and reaction type[d]	Days to full flag leaf[e]	Plant height (cm)	Head type[f]	Growth habit[g]
223	*T. turgidum* ssp. *dicoccoides*	3⁺	1ᶜ	40MR-MS	172	107	8	B
224	*T. turgidum* ssp. *dicoccoides*	3⁺	2	40MS	172	92	8	B
225	*T. turgidum* ssp. *dicoccoides*	3	3ᶜ	70MS-S	160	82	8	B
226	*T. turgidum* ssp. *dicoccoides*	2	2⁺	40MS	77	90	8	C
227	*T. turgidum* ssp. *dicoccoides*	3	2⁺⁺ᶜ	50MS	187	111	8	B
228	*T. turgidum* ssp. *dicoccoides*	3	3ᶜ	60MS	187	117	8	B
229	*T. turgidum* ssp. *dicoccoides*	3⁺	2⁻	50MS	172	116	8	B
230	*T. turgidum* ssp. *dicoccoides*	3⁺	4	80S	75	136	8	B
231	*T. turgidum* ssp. *dicoccoides*	3⁺	4	80S	69	138	8	B
232	*T. turgidum* ssp. *dicoccoides*	3	3⁺	80S	69	119	8	B
233	*T. turgidum* ssp. *dicoccoides*	3⁺	4	80S	75	124	8	B
234	*T. turgidum* ssp. *dicoccoides*	3⁺	4	80S	77	112	8	B
235	*T. turgidum* ssp. *dicoccoides*	3⁺	4	80S	75	118	8	B
236	*T. turgidum* ssp. *dicoccoides*	3	4	80S	75	70	8	B
237	*T. turgidum* ssp. *dicoccoides*	3	4	80S	69	127	8	B
238	*T. turgidum* ssp. *dicoccoides*	3⁺⁺	4	80S	75	134	8	B
239	*T. turgidum* ssp. *dicoccoides*	3⁺	4	80S	69	140	8	B
240	*T. turgidum* ssp. *dicoccoides*	3⁺	4	80S	75	122	8	B
241	*T. turgidum* ssp. *dicoccoides*	3⁺	4	80S	69	121	8	B
242	*T. turgidum* ssp. *dicoccoides*	3⁺⁺	4	80S	75	132	8	B

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Table 2 (cont.). Evaluation of a collection of wild *Triticum* species for resistance to *Puccinia recondita* f. sp. *tritici*[a] and criteria used to differentiate among accessions

Accession Number	Species[b]	Seedling infection type[c]	Adult plant infection type[c]	Adult plant leaf rust severity and reaction type[d]	Days to full flag leaf[e]	Plant height (cm)	Head type[f]	Growth habit[g]
243	*T. turgidum* ssp. *dicoccoides*	3	4	80S	69	132	8	B
244	*T. turgidum* ssp. *dicoccoides*	3	3	80MS-S	77	80	8	B
245	*T. turgidum* ssp. *dicoccoides*	3⁺	4	80S	69	141	8	B
246	*T. turgidum* ssp. *dicoccoides*	3⁺	4	80S	69	120	8	B
247	*T. turgidum* ssp. *dicoccoides*	3	4	80S	75	134	8	B
248	*T. turgidum* ssp. *dicoccoides*	3	4	80S	69	136	8	B
249	*T. turgidum* ssp. *dicoccoides*	3	4	80S	69	130	8	C
250	*T. turgidum* ssp. *dicoccoides*	3⁺⁺	4	80S	75	103	8	B
251	*T. turgidum* ssp. *dicoccoides*	3⁺⁺	4	80S	75	131	8	B
252	*T. turgidum* ssp. *dicoccoides*	3⁺	4	80S	89	72	8	B
253	*T. turgidum* ssp. *dicoccoides*	3⁺	2	50MS	118	106	8	B
254	*T. turgidum* ssp. *dicoccoides*	3⁺⁺	3⁺	50S	132	83	8	B
255	*T. turgidum* ssp. *dicoccoides*	3	3⁻ᶜ	30MS-S	125	83	8	B
256	*T. turgidum* ssp. *dicoccoides*	3	3	30S	132	84	8	B
257	*T. turgidum* ssp. *dicoccoides*	3⁺	3	60MS-S	125	104	8	B
258	*T. turgidum* ssp. *dicoccoides*	3⁺⁺	3ᶜ	60MS	125	66	8	B
259	*T. turgidum* ssp. *dicoccoides*	3⁺	4	70S	125	106	8	B
260	*T. turgidum* ssp. *dicoccoides*	3⁺	3⁺	60S	125	104	8	B
261	*T. turgidum* ssp. *dicoccoides*	3⁺	3ᶜ	50MS-S	125	77	8	B
262	*T. turgidum* ssp. *dicoccoides*	3⁺	3⁺	50S	132	111	8	B

Table 2 (cont.). Evaluation of a collection of wild *Triticum* species for resistance to *Puccinia recondita* f. sp. *tritici*[a] and criteria used to differentiate among accessions

Accession Number	Species[b]	Seedling infection type[c]	Adult plant infection type[c]	Adult plant leaf rust severity and reaction type[d]	Days to full flag leaf[e]	Plant height (cm)	Head type[f]	Growth habit[g]
263	*T. turgidum* ssp. *dicoccoides*	2,3	2	50MS	118	106	8	B
264	*T. turgidum* ssp. *dicoccoides*	3*	3*c	70MS-S	125	93	8	B
265	*T. turgidum* ssp. *dicoccoides*	3	2*c	70MS	153	97	8	B
266	*T. turgidum* ssp. *dicoccoides*	;13	4	80S	54	95	10	B
267	*T. turgidum* ssp. *dicoccoides*	3	2**	40MR-MS	118	89	8	B
268	*T. turgidum* ssp. *dicoccoides*	3	2	40MS	132	74	8	B
269	*T. turgidum* ssp. *dicoccoides*	2-	2**	30MS	153	69	8	B
270	*T. turgidum* ssp. *dicoccoides*	3*	4	80S	111	54	8	B
271	*T. turgidum* ssp. *dicoccoides*	3**	3*	50S	132	76	8	B
272	*T. turgidum* ssp. *dicoccoides*	3**	4	80S	118	102	8	B
273	*T. turgidum* ssp. *dicoccoides*	3	2**	40MS-S	153	78	8	B
275	*T. turgidum* ssp. *dicoccoides*	3	2-	30MR-MS	172	77	8	B
276	*T. turgidum* ssp. *dicoccoides*	2,3	3	60S	153	74	8	B
277	*T. turgidum* ssp. *dicoccoides*	2	2c	40MS	187	78	8	B
278	*T. turgidum* ssp. *dicoccoides*	3*	4	80S	63	106	8	B
279	*T. turgidum* ssp. *dicoccoides*	3*	4	80S	89	70	8	B
280	*T. turgidum* ssp. *dicoccoides*	3*	3**	60S	132	94	8	B
281	*T. turgidum* ssp. *dicoccoides*	3*	2-	50MS	172	90	8	B
282	*T. turgidum* ssp. *dicoccoides*	3*	3**	80S	125	93	8	B
283	*T. turgidum* ssp. *dicoccoides*	3	3-	60MS-S	125	74	8	B

Table 2 (cont.). Evaluation of a collection of wild *Triticum* species for resistance to *Puccinia recondita* f. sp. *tritici*[a] and criteria used to differentiate among accessions

Accession Number	Species[b]	Seedling infection type[c]	Adult plant infection type[c]	Adult plant leaf rust severity and reaction type[d]	Days to full flag leaf[e]	Plant height (cm)	Head type[f]	Growth habit[g]
284	*T. turgidum* ssp. *dicoccoides*	3[++]	4	80S	118	82	8	B
285	*T. turgidum* ssp. *dicoccoides*	3	3[++]	70S	153	103	8	B
286	*T. turgidum* ssp. *dicoccoides*	3	3[+]	50S	132	84	8	B
287	*T. turgidum* ssp. *dicoccoides*	3[+]	4	80S	118	91	8	B
288	*T. turgidum* ssp. *dicoccoides*	3	4	80S	111	67	8	B
289	*T. turgidum* ssp. *dicoccoides*	3	4	70S	125	78	8	B
290	*T. turgidum* ssp. *dicoccoides*	3	4	80S	89	106	8	B
291	*T. turgidum* ssp. *dicoccoides*	3[+]	3[-]	60MS	118	52	8	B
292	*T. turgidum* ssp. *dicoccoides*	3[+]	3	60MS-S	118	81	8	B
293	*T. turgidum* ssp. *dicoccoides*	3[-]	3[-c]	50MR-MS	104	88	8	B
294	*T. turgidum* ssp. *dicoccoides*	3	2	40MR-MS	118	92	8	B
295	*T. turgidum* ssp. *dicoccoides*	3[+]	3	50S	77	110	8	B
296	*T. turgidum* ssp. *dicoccoides*	;1,2	3	60MS-S	111	96	8	B
297	*T. turgidum* ssp. *dicoccoides*	3[++]	3	60MS-S	111	82	8	B
298	*T. turgidum* ssp. *dicoccoides*	2	4	80S	89	94	8	B
299	*T. turgidum* ssp. *dicoccoides*	2	3	70MS-S	111	103	8	B
300	*T. turgidum* ssp. *dicoccoides*	3	3	60MS-S	118	93	8	B
301	*T. turgidum* ssp. *dicoccoides*	;1	4	80S	111	86	8	B
302	*T. turgidum* ssp. *dicoccoides*	2,3	3[-]	60S	104	106	8	B
306	*T. turgidum* ssp. *dicoccoides*	3[++]	3[+]	60S	172	106	8	B

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Table 2 (cont.). Evaluation of a collection of wild *Triticum* species for resistance to *Puccinia recondita* f. sp. *tritici*[a] and criteria used to differentiate among accessions

Accession Number	Species[b]	Seedling infection type[c]	Adult plant infection type[c]	Adult plant leaf rust severity and reaction type[d]	Days to full flag leaf[e]	Plant height (cm)	Head type[f]	Growth habit[g]
308	*T. turgidum* ssp. *dicoccoides*	3[+]	2[c]	30MR-MS	132	84	8	B
317	*T. turgidum* ssp. *dicoccoides*	3	3	60S	63	142	8	B
320	*T. turgidum* ssp. *dicoccoides*	2,3	2[c]	60MS	75	103	8	C
346	*T. turgidum* ssp. *dicoccoides*	3[+]	2[c]	70MS	63	115	10	B
358	*T. turgidum* ssp. *dicoccoides*	3[+]	3	70S	54	101	10	B
366	*T. turgidum* ssp. *dicoccoides* var. fulvo-villosum	2[c]	2[−]	60MR-MS	75	95	10	B
377	*T. turgidum* ssp. *dicoccoides*	3[+]	3	30S	153	106	8	B
378	*T. turgidum* ssp. *dicoccoides*	3[++]	4	80S	89	92	8	B
379	*T. turgidum* ssp. *dicoccoides*	;1[+]	2[c]	40MS	132	96	8	B
380	*T. turgidum* ssp. *dicoccoides*	3[+]	;1	20MR	172	98	8	B
381	*T. turgidum* ssp. *dicoccoides*	3	2	40MR-MS	132	82	8	B

[a] Leaves were inoculated with a mixture of pathotypes UVPRT2,3,9 and 13.

[b] Species nomenclature according to germplasm collection of the Departement of Genetics, University of Stellenbosch.

[c] Infection types were scored according to a 0 to 4 scale where 0=absence of macroscopic symptoms and 4=susceptibility; x = resistant and z = susceptible. Flecks are indicated by ; and chlorosis and necrosis by "C" and "N", respectively. Plus or minus signs indicate variation above or below established pustule sizes. Infection types 23 indicate a range of 2 and 3 reactions on the same leaf and ;13 a range between ;1 and 3. Infection type 2,3 indicates within-line variation for leaf rust reaction.

[d] Adult plant reaction type was measured on a 0 to 100 scale. Resistant (R), trace resistant (tR), moderately resistant (MR), moderately susceptible (MS) and susceptible (S) reaction types are also indicated.

[e] Days from planting until full emergence of flag leaf.

[f] Head type were characterized according to Figure 1.

[g] Growth habit were classified as either prostrate (A), intermediate (B) or erect (C).

[h] No ears emerged.

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Table 3. A summary of the reaction of accessions tested both at seedling and adult stages, to pathotypes UVPrt 2, 3, 9 and 13 of *Puccinia recondita* f. sp. *tritici*

Species	No. of accessions in each species	Seedlings			Adult plants		
		R[a]	I[b]	S[c]	R[a]	I[b]	S[c]
T. tauschii	17	1	14	2	11	2	4
T. timopheevii	3	1	2	0	2	1	0
T. timopheevii ssp. *araraticum*	3	0	1	2	3	0	0
T. timopheevii ssp. *ararativum* var. tumanianii	5	0	2	3	3	0	2
T. timopheevii ssp. *araraticum* var. nachitchevanicum	9	0	3	6	5	1	3
T. bicorne	9	0	1	8	2	1	6
T. bicorne var. typicum	1	0	0	1	0	0	1
T. cylindricum	2	0	1	1	0	1	1
T. juvenale	4	0	1	3	1	0	3
T. kotschyi	3	1	2	0	3	0	0
T. longissimum	7	5	1	1	7	0	0
T. sharonense	6	1	3	2	6	0	0
T. sharonense var. typicum	1	1	0	0	1	0	0
T. searsii	5	2	2	1	5	0	0
T. ventricosum	2	0	2	0	2	0	0
T. syriacum	3	0	3	0	1	2	0
T. peregrinum	1	1	0	0	1	0	0
T. turgidum var. nigro-barbatum	1	0	0	1	1	0	0

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Table 3 (cont.). A summary of the reaction of accessions tested both at seedling and adult stages, to pathotypes UV*Prt* 2, 3, 9 and 13 of *Puccinia recondita* f. sp. *tritici*

Species	No. of accessions in each species	Seedlings			Adult plants		
		R[a]	I[b]	S[c]	R[a]	I[b]	S[c]
T. turgidum ssp. *dicoccum* var. arras	2	1	1	0	1	0	1
T. turgidum ssp. *dicoccum* var. farrum	2	1	1	0	1	0	1
T. turgidum ssp. *dicoccum*	2	0	2	0	2	0	0
T. turgidum ssp. *dicoccum* var. rufum	3	3	0	0	2	1	0
T. turgidum ssp. *dicoccum* var. khapli	1	1	0	0	0	0	1
T. turgidum ssp. *anyleum* var. atratum	1	0	0	1	1	0	0
T. turgidum ssp. *anyleum* var. rufum	3	1	2	0	2	1	0
T. turgidum ssp. *anyleum* var. tricoccum	1	0	0	1	0	1	0
. turgidum ssp. *anyleum* var. nigro-ajar	1	1	0	0	1	0	0
T. turgidum ssp. *anyleum* var. macroanterum	1	0	0	1	1	0	0
T. turgidum ssp. *anyleum*	1	0	1	0	1	0	0
T. turgidum ssp. *abyssinicum*	1	1	0	0	1	0	0
T. turgidum ssp. *palaeocolchicum* var. schwamilicum	2	0	2	0	2	0	0
T. turgidum ssp. *palaeocolchicum*	3	0	3	0	3	0	0
T. turgidum ssp. *durum* var. reichenbachii	1	1	0	0	1	0	0
T. turgidum ssp. *durum* var. hordeiforme [a]	1	1	0	0	1	0	0
T. turgidum ssp. *durum* var. melanopus	1	0	1	0	1	0	0
T. turgidum ssp. *durum* var. africanum	1	0	0	1	0	0	1
T. turgidum ssp. *durum* var. murciense	1	0	1	0	1	0	0

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Table 3 (cont.). A summary of the reaction of accessions tested both at seedling and adult stages, to pathotypes UV*Prt* 2, 3, 9 and 13 of *Puccinia recondita* f. sp. *tritici*

Species	No. of accessions in each species	Seedlings			Adult plants		
		R[a]	I[b]	S[c]	R[a]	I[b]	S[c]
T. turgidum ssp. *durum* var. obscurum	1	0	1	0	1	0	0
T. turgidum ssp. *durum* var. aestivum	1	1	0	0	1	0	0
T. turgidum ssp. *durum* var. duro-compactum	1	1	0	0	0	0	1
T. turgidum ssp. *durum* var. libycum	1	0	1	0	1	0	0
T. turgidum ssp. *persicum* var. stramineum	5	0	1	4	3	1	1
T. turgidum ssp. *persicum* var. fuliginosum	1	0	0	1	0	1	0
T. turgidum ssp. *persicum* var. rubiginosum	2	0	0	2	1	1	0
T. turgidum ssp. *persicum*	7	0	4	3	0	4	3
T. turgidum ssp. *pyramidale* var. recognitum	2	2	0	0	2	0	0
T. turgidum ssp. *pyramidale* var. compacticum	1	0	0	1	0	0	1
T. turgidum ssp. *pyramidale*	2	1	1	0	2	0	0
T. turgidum ssp. *compactum*	2	2	0	0	2	0	0
T. turgidum ssp. *orientale* var. insigne	2	0	0	2	0	0	2
T. turgidum ssp. *orientale* var. notabile	2	0	0	2	1	0	1
T. turgidum ssp. *orientale* var. euisgne	1	0	1	0	1	0	0
T. turgidum ssp. *aethiopicum*	2	1	1	0	1	0	1
T. turgidum ssp. *aethiopicum* var. atratosanquineum	1	0	0	1	0	1	0
T. turgidum ssp. *polonicum* var. vesticum	2	0	1	1	2	0	0
T. turgidum ssp. *polonicum* var. levissimum	1	0	1	0	0	1	0

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Table 3 (cont.). A summary of the reaction of accessions tested both at seedling and adult stages, to pathotypes UV*Prt* 2, 3, 9 and 13 of *Puccinia recondita* f. sp. *tritici*

Species	No. of accessions in each species	Seedlings			Adult plants		
		R[a]	I[b]	S[c]	R[a]	I[b]	S[c]
T. turgidum ssp. *polonicum*	4	0	3	1	2	0	2
T. turgidum ssp. *isphahanicum*	1	0	0	1	1	0	0
T. turgidum ssp. *carthlicum*	1	0	0	1	0	0	1
T. turgidum ssp. *carthlicum* var. *stramineum*	1	0	1	0	0	1	0
T. turgidum ssp. *dicoccoides*	179	2	20	157	19	39	121
T. turgidum ssp. *dicoccoides* var. kotschianum	5	0	0	5	1	0	4
T. turgidum ssp. *dicoccoides* var. spontaneonigrum	2	0	1	1	0	0	2
T. turgidum ssp. *dicoccoides* var. straussianum	1	0	0	1	1	0	0
T. turgidum ssp. *dicoccoides* var. arabicum	1	0	0	1	0	0	1
T. turgidum ssp. *dicoccoides* var. spontaneovillosum	2	0	0	2	0	1	1
T. turgidum ssp. *dicoccoides* var. aaronsohni	3	0	0	3	0	0	3
T. turgidum ssp. *dicoccoides* var. fulvo-villosum	4	0	2	2	0	2	2
T. turgidum ssp. *dicoccoides* var. pseudorufovillosum	1	0	1	0	0	1	0
	353	34	92	227	118	64	171

[a] Infection types X, 0, 1, Z and ; were classified as resistant (R).
[b] Infection type 2 was classified as intermediate (I).
[c] Infection types 3 and 4 were classified as susceptible (S).

Fig. 1. Head types (1-5) of the different *Triticum* species (Peterson, 1965; Miller, 1987).
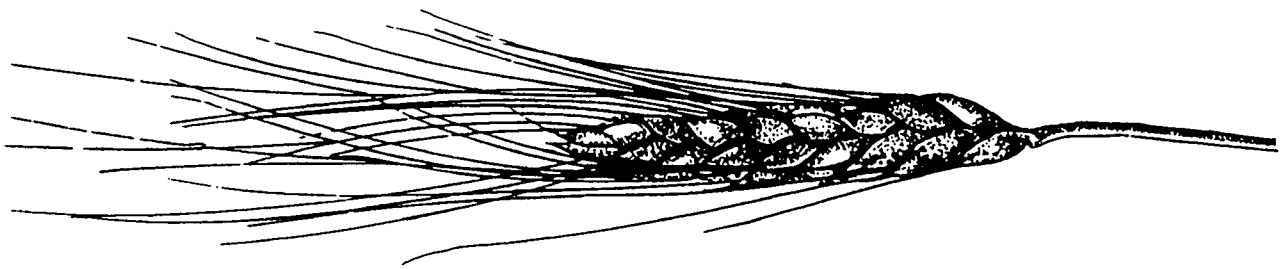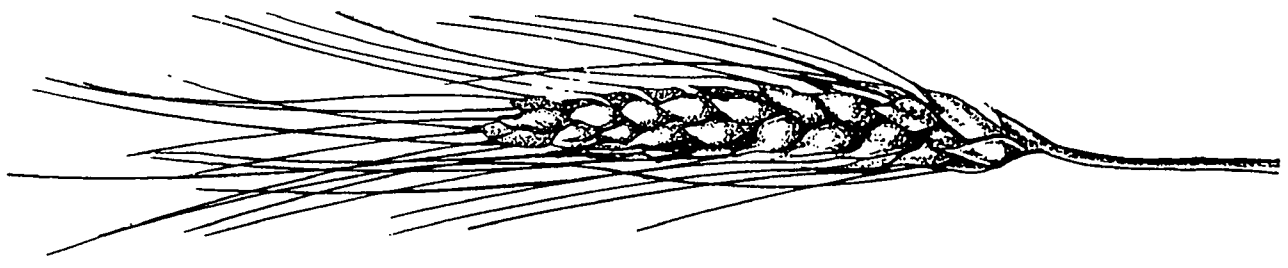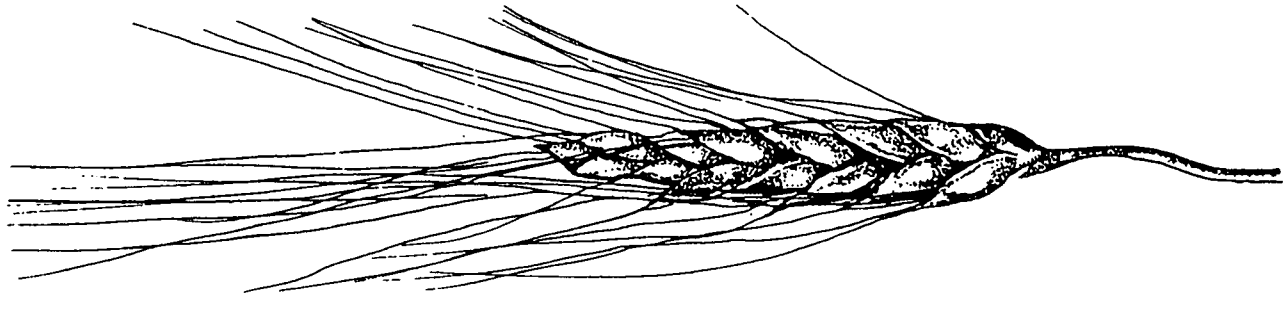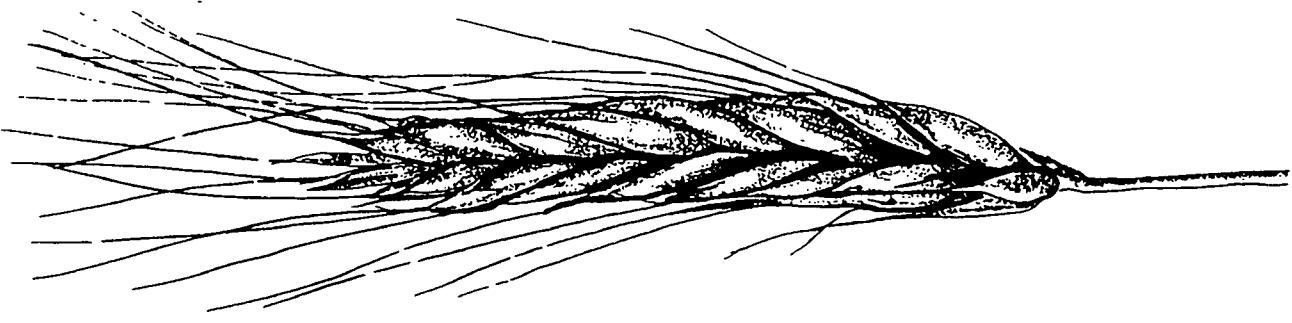
1   2   3   4   5

Fig. 1 (cont). Head types (6-10) of the different *Triticum* species (Peterson, 1965; Miller, 1987).

6   7   8   9   10

Fig. 1 (cont). Head types (11-13) of the different *Triticum* species (Peterson, 1965; Miller, 1987).

11   12   13
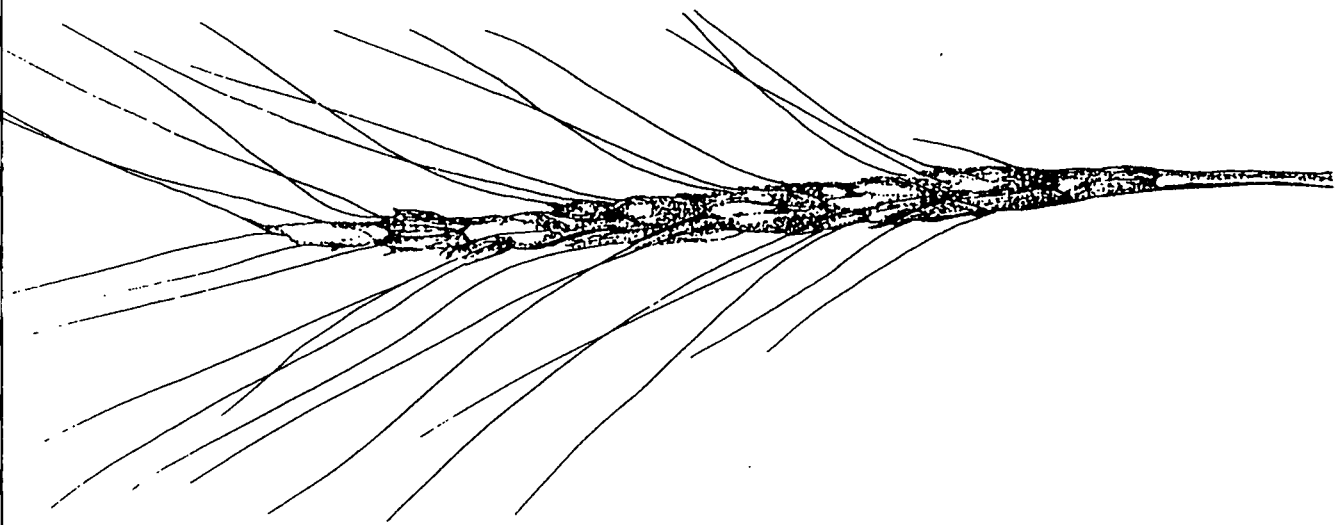
Fig. 1 (cont). Head types (14-16) of the different *Triticum* species (Peterson, 1965; Miller, 1987).

14　　15　　16
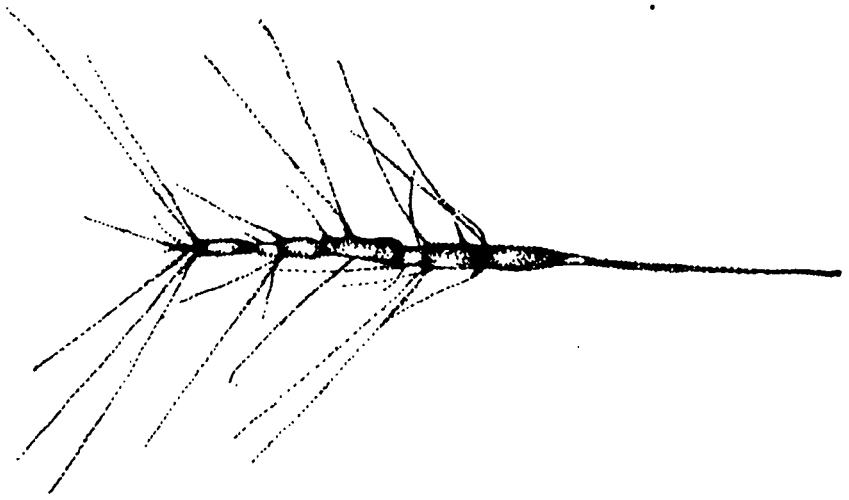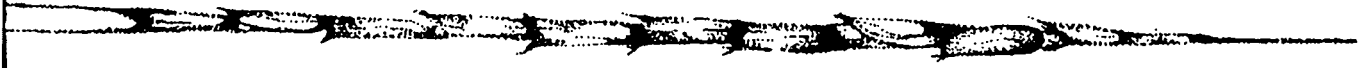
Fig. 2. Infection types produced by a mixture of pathotypes UVPrt 2, 3, 9 and 13 of *Puccinia recondita* f. sp. *tritici* on flag leaves of (A) *Triticum longissimum*, (B) *T. tauschii*, (C) *T. timopheevii* and (D) *T. turgidum* ssp. *dicoccoides*.
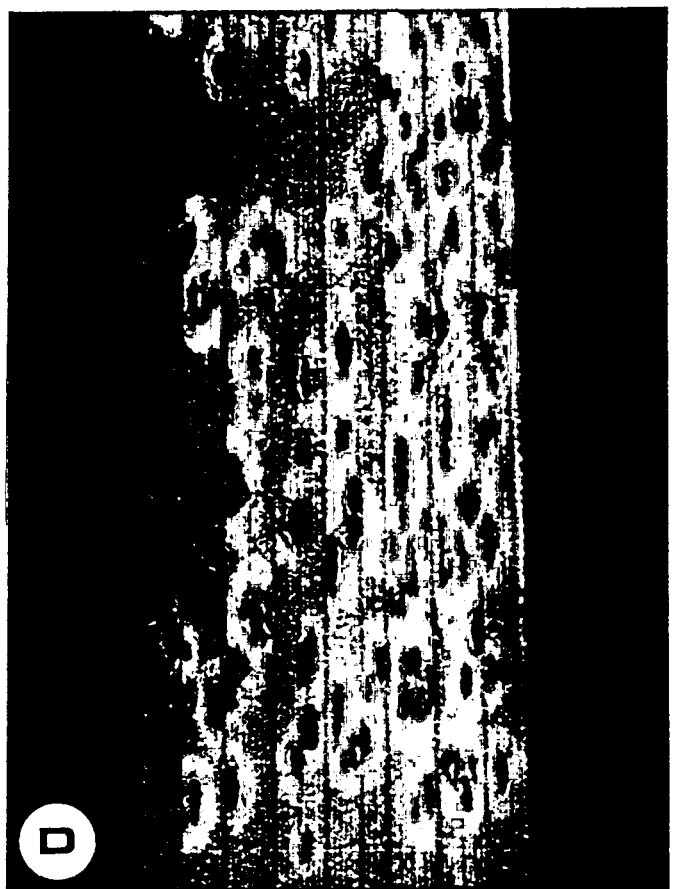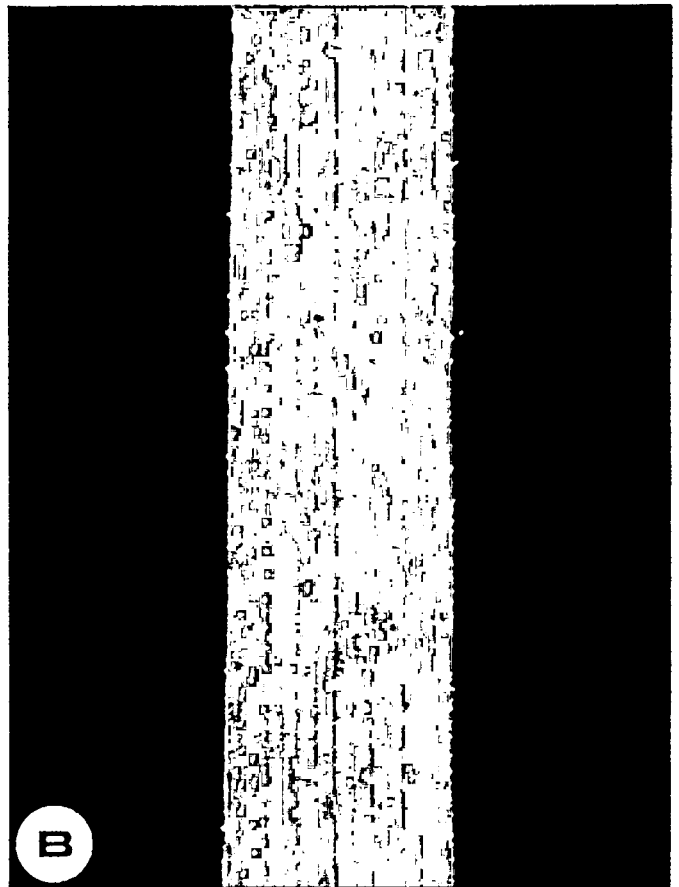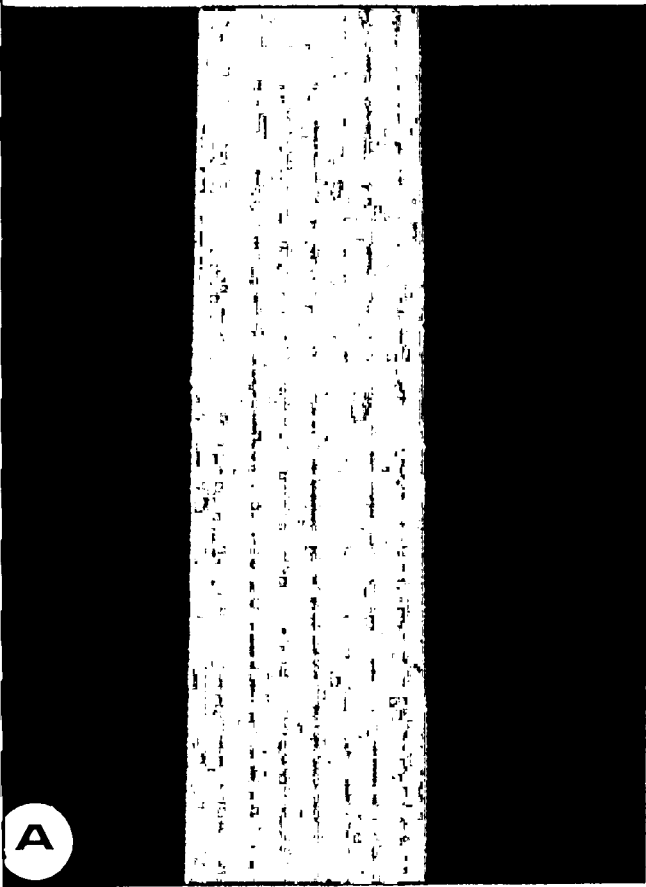
CHAPTER 3

PARTIAL RESISTANCE TO *PUCCINIA RECONDITA*
f. sp. *TRITICI* IN SELECTED *TRITICUM* SPECIES

ABSTRACT

The aim of this study was to quantify components of resistance to leaf rust in certain *Triticum* species. Accessions selected were previously shown to produce smaller or fewer leaf rust pustules without the characteristic chlorosis and necrosis associated with hypersensitive resistance. Latent period of leaf rust, uredium size and density, and infection type were determined in two experiments on flag leaves of 13 accessions of *T. turgidum, T. timopheevii* and *T. tauschii.* Plants were quantitatively inoculated with pathotype UVPrt13 of *P. recondita* f. sp. *tritici.* Palmiet, a bread wheat cultivar susceptible to UVPrt13, was included as a control. In the first experiment latent period ranged from 309 h to 401 h compared to 258 h in the susceptible control, Palmiet. In the second experiment Palmiet had a latent period of 244 h whereas those in the *Triticum* accessions ranged between 175 h and 372 h. Most accessions supported more uredia per cm^2 flag leaf surface than Palmiet in the first, but not in the second experiment. However, pustules were significantly smaller on most of the lines. Based on these components, *T. timopheevii* ssp. *araraticum* var. tumanianii, *T. turgidum* ssp. *durum* var. obscurum, *and T. turgidum* ssp. *persicum* var. stramineum, showed high levels of partial resistance. Transfer of such resistance to commercial wheat varieties should contribute to long-lasting genetic control of leaf rust.

INTRODUCTION

Leaf rust, caused by *P. recondita* Rob. ex. Desm. f. sp. *tritici* (Eriks. & Henn), is one of the most destructive and widely distributed diseases of wheat. Changes in virulence in the leaf rust pathogen have been frequent (Samborski, 1982; Statler *et al.*, 1982; Bennett, 1984; Pretorius, 1988) and in most wheat growing areas of the world, distressingly few genes remain that provide useful levels of leaf rust resistance (Browder, 1980; Bennett, 1984; Kolmer, 1996).

Wild relatives of common wheat (*T. aestivum* L.) represent a large and important

pool of genetic variation for broadening and diversifying disease resistance. There have been several successful transfers of genes for resistance to leaf rust from species related to bread wheat (Knott, 1979; Browder, 1980; Sharma & Gill, 1983; Dyck & Kerber, 1985; Bai *et al.*, 1993; Cox *et al.*, 1994; Davoyan & Ternovskaya, 1996; Brown-Guedira *et al.*, 1997), specifically from the wild diploid species *T. tauschii* (syn. *Aegilops squarrosa*) (Dyck & Kerber, 1970; Rowland & Kerber, 1974; Kerber, 1987; Gill et *al.*, 1988; Kerber & Dyck, 1990; Gill *et al.*, 1991; Cox *et al.*, 1992a; Cox *et al.*, 1992b; Cox *et al.*, 1994). Landraces have also been shown to be useful sources of resistance (Gill *et al.*, 1986; Negassa, 1987; Manisterski *et al.*, 1988; Cox *et al.*, 1992a; Singh *et al.*, 1992; The *et al.*, 1993; Antonov & Marais, 1996). In view of the few effective genes available, it is imperative that studies searching for new resistance genes continue.

In wheat, the resistance phenotype of leaf rust varies from a hypersensitive response (HR) to partial resistance (PR). Resistance classified as HR may vary considerably in expression. The low infection type resulting from the HR is characterised by collapsed host cells, which often are visible as necrotic flecks or severe necrosis and/or necrosis associated with small to intermediate pustules. The gene *Lr37*, derived from *Ae. ventricosa*, is an example of a gene mediating HR in adult plants to leaf rust (Dyck and Lukow, 1988; Pretorius, 1990). Partial resistance (slow rusting) is expressed by a susceptible host reaction but slower rate of disease development (Lee & Shaner, 1985a; Broers & De Haan, 1994). Components of resistance in slow rusting cultivars are longer latent periods, smaller and fewer uredia, and reduced spore production (Ohm & Shaner, 1976; Das *et al.*, 1993). In the case of PR no cell collapse occurs and this resistance type is considered to be durable (Parlevliet, 1975b; Parlevliet, 1978).

Preliminary disease assessments in a collection of *Triticum* species, identified several accessions with PR to leaf rust (Chapter 2). The aim of this study was to quantify components of resistance to leaf rust, using controlled inoculation techniques, in some of those species.

MATERIALS & METHODS

Growing of _Triticum_ species Ten accessions of _T. turgidum_ L., two of _T. timopheevii_ (Zhuk.) Zhuk., one of _T. tauschii_ (Coss.) Schmal. (Table 1), and the susceptible bread wheat cultivar Palmiet, were grown in a leaf rust-free, air-conditioned glasshouse cubicle. To investigate the consistency of results, two similar, replicated experiments were conducted. Three plants were grown per 1-L-capacity pot. During the two experiments day/night temperature variation of $25.2\pm2.2°C/15.3\pm0.26°C$, and $24\pm1.54°C/14.5\pm0.43°C$, were recorded, respectively. Natural daylight was supplemented with 120 μmol m^{-2}s^{-1} photosynthetically active radiation emitted by cool-white fluorescent tubes, arranged directly above plants, for 14 h each day. Seven days after planting, and three times weekly thereafter, 50 ml of 3g/L hydroponic nutrient solution (6.5:2.7:13 N:P:K plus micro-elements), were added as a soil drench per pot. Fertilisation continued until the experiment was terminated.

Inoculation Flag leaves were quantitatively inoculated with freshly harvested spores of pathotype UVPrt13 of _P. recondita_ f. sp. _tritici_, suspended in light mineral oil. According to infection types produced on the South African differential set, this pathotype is avirulent to the leaf rust resistance genes _Lr3a, 3bg, 3ka, 11, 16, 20, 30_ and virulent to _Lr1, 2a, 2b, 2c, 10, 14a, 15, 17, 24_ and _26_ (Pretorius _et al._, 1995). UVPrt13, one of the most commonly occurring leaf rust pathotypes in South Africa, is virulent on Palmiet used as a susceptible control in this experiment. A 3-cm-long area, halfway between the leaf base and tip, on the adaxial flag leaf surface of each of eight main tillers per genotype, was marked and inoculated using a modified, vertically spraying device (Andres & Wilcoxson, 1984; Kloppers & Pretorius, 1995). Using lacquer-coated glass slides, calibration of the inoculation device revealed that 133±17 spores were deposited per square centimetre in the first experiment and 129±20 in the second experiment. The viability of urediospores was determined by microscopic examination of the germination of the spore suspension on 2% water agar plates, incubated in the dark for 3 h at 20°C. A minimum of 99% urediospore germination was observed in both experiments. After inoculation, plants were kept in the dark in a dew-simulation chamber at 21-22°C for 16 h. Upon removal from the chamber, plant

surfaces were allowed to dry for 2 h in fan-circulated air before plants were transferred to a 6.5 m^2 air-conditioned glasshouse cubicle and maintained in conditions similar to those described for the pre-inoculation period.

Components of resistance Three components of resistance, namely latent period, uredium size and uredium density, were quantified.

Latent period A designated portion on each inoculated leaf was inspected daily and the number of uredia visible as erumpent structures recorded. These counts continued until the exponential phase of primary uredial appearance had passed. A final count was made two days later. The latent period was estimated, using linear regression of log-transformed uredial counts against time, as the number of hours after inoculation when 40% of the uredia were visible (Andres, 1982).

Uredium density When final counts had been made and IT's determined, inoculated leaves were sampled. The area of the portion on which uredia had been counted on for each leave was determined using a leaf area meter (model CI-251, CID Inc., Moscow, Idaho, USA). The uredium density was calculated as the number of uredia per centimetre square leaf area.

Uredium size Four leaves from each treatment were photographed at known magnification on colour slide film. From projected slide images, uredium size was determined by measuring the length and width of 10 non-coalescing pustules per leaf with a digital micrometer and calculating the areas (mm^2) according to the formula π x length x width/4.

Infection types Disease reactions were rated according to a 0 to 4 infection type (IT) scale (Roelfs, 1988b) 21 d.p.i. (experiment 1) and 23 d.p.i. (experiment 2) on flag leaves of plants.

Statistical analysis Analysis of variance was done with SOLO (BMDP Statistical Software Inc., Los Angeles, CA), using the procedure for a general linear model. Standard deviations were calculated to compare means. For each component, data from the first and second experiments were combined if experiment-by-treatment interaction was not significant, and if experiments, according to analysis of variance,

were similar.

RESULTS

Infection types recorded on the different species are presented in Table 1. Low IT's (1⁻ to 1⁺⁺) were observed on flag leaves of *T. turgidum* ssp. *carthlicum* var. stramineum, *T. tauschii*, *T. turgidum* ssp. *durum* var. obscurum and *T. turgidum* ssp. *persicum* var. stramineum. *Triticum timopheevii* ssp. *araraticum* var. tumanianii. *T. turgidum* ssp. *dicoccum* var. rufum, *T. turgidum* ssp. *anyleum* var. rufum, *T. timopheevii*, *T. turgidum* ssp. *pyramidale* var. recognitum (I) and *T. turgidum* ssp. *pyramidale* produced intermediate IT's (2 to 2⁺⁺). Susceptible IT's (3 to 3⁺⁺) were observed in *T. turgidum* ssp. *dicoccoides*, *T. turgidum* ssp. *pyramidale* var. recognitum (II) and *T. turgidum* ssp. *pyramidale* var. compiticum. Infection types agreed with previous results (Chapter 2), except for *T. turgidum* ssp. *carthlicum* var. stramineum (2⁺), *T. turgidum* ssp. *pyramidale* var. recognitum (I) (1), *T. turgidum* ssp. *pyramidale* (1) and *T. turgidum* ssp. *pyramidale* var. recognitum (II) (1).

Latent period Latent period of leaf rust was significantly ($P<0.05$) influenced by host genotype and experiment. The two experiments differed significantly with latent periods in the second experiment being shorter than in the first (Fig. 1). In the first experiment, latent period in the accessions ranged from 309±15 h to 401±10 h compared to 258±12 h in the susceptible control, Palmiet (Fig. 1A). Latent period was most extended (401 h) in *T. timopheevii*. In the second experiment, a latent period of 244±8 h was recorded in Palmiet whereas the *Triticum* accessions ranged between 175±13 h and 372±11 h (Fig. 1B). In this experiment, shorter latent periods were recorded in *T. turgidum* ssp. *anyleum* var. rufum, *T. tauschii*, *T. turgidum* ssp. *pyramidale* var. recognitum (I) and *T. turgidum* ssp. *pyramidale* var. recognitum (II) than in the control.

Uredium density The number of uredia was significantly ($P<0.05$) influenced by host genotype and experiment. In experiment one (Figure 2A) only *T. timopheevii* ssp. *araraticum* var. tumanianii, *T. turgidum* ssp. *durum* var. obscurum and *T. turgidum* ssp. *persicum* var. stramineum supported less uredia per square centimeter leaf area than

the control. In the second experiment all accessions had lower uredium densities than the control except *T. tauschii* and *T. turgidum* ssp. *pyramidale* var. compiticum (Fig. 2B).

Uredium size The mean size of uredia (Fig. 3) did not differ significantly between the two experiments, but was significantly influenced by host genotype. Uredia produced on all accessions, except *T. turgidum* ssp. *pyramidale*, *T. turgidum* ssp. *pyramidale* var. recognitum (II) and *T. turgidum* ssp. *pyramidale* var. compiticum, were smaller than those on Palmiet.

DISCUSSION

Variation observed in IT's between previous experiments (Chapter 2) and this study can probably be ascribed to the fact that a pathotype mixture was used earlier compared to pathotype UVPrt13 in this study.

Based on the three components measured and their comparison with Palmiet, *T. timopheevii* ssp. *araraticum* var. tumanianii, *T. turgidum* ssp. *durum* var. obscurum, and *T. turgidum* ssp. *persicum* var. stramineum, showed high levels of PR. If the relatively high spore densities are taken into account, *T. turgidum* ssp. *dicoccum* var. rufum, *T. timopheevii*, *T. turgidum* ssp. *dicoccoides* and *T. turgidum* ssp. *carthlicum* var. stramineum could also be viewed as partially resistant. Although PR, characterised by a longer latent period, and fewer and smaller uredia, has the best potential of restricting rust development in field epidemics, these components are not necessarily linked. Sometimes, the effect of PR is reduced due to environmental conditions (Broers & Parlevliet, 1989). Pretorius *et al*. (1987b) found that the *Lr22a* gene for APR to leaf rust produced a slow rusting response typified by a long latent period and small uredia. However, uredium density was not reduced. In cereal rust host-pathogen systems, previous studies (Broers & Wallenburg, 1989; Drijepondt & Pretorius, 1989; Jacobs & Kiriswa, 1993; Pretorius *et al*., 1994) indicated that the individual components have not always been dependable to describe resistance. Apparently, the sensitivity in expression of components is due to host rather than pathogen factors (Broers & Wallenburg, 1989).

Slow rusting resistance in alien species has not been evaluated as extensively

as hypersensitivity, partly because it is more difficult to work with. The majority of APR genes described in alien species (Knott, 1987; Dyck & Lukow, 1988) have been race-specific genes (Jones et al., 1995). This report, together with previous results, (Statler et al., 1977; Wilson & Shaner, 1987) suggest that alien species, especially those with chromosomes homologous to those in hexaploid wheat, could also be useful sources of slow rusting. Wilson & Shaner (1987) indicated that triticale may be a useful donor of leaf rust resistance contributing genes for both hypersensitivity and slow rusting. Statler et al. (1977) identified slow rusting in tetraploid durum wheat which they thought would confer acceptable resistance to leaf rust.

Several aspects need to be investigated further before the resistance identified in this study can be utilised. In the present study only one cycle of infection was investigated. A better understanding of the resistance will be obtained in field evaluations where the materials, including appropriate control entries, are exposed to high disease pressure in artificially created epidemics. By using a mixture of pathotypes in these experiments, the stability of resistance to pathogenic variability could also be determined. A comparison of the respective area under the disease progress curves will prove useful in detecting PR.

When leaf rust resistance genes are transferred from diploid to hexaploid wheats, the degree of resistance decreases with increasing levels of ploidy (Kerber & Dyck, 1969; Kerber & Dyck, 1973). Since the partially resistant T. timopheevii ssp. araraticum var. tumanianii, T. turgidum ssp. durum var. obscurum, and T. turgidum ssp. persicum var. stramineum are tetraploid, further studies on the possible dilution of resistance in a bread wheat background are needed. The transfer of resistance genes from related species of lower-ploidy into hexaploid bread wheat can also be complicated by interactions between resistance genes and suppressor genes in the different genomes (Kolmer, 1996). Bai & Knott (1992) found that F_1 plants of crosses between T. aestivum (AABBDD) and accessions of T. turgidum ssp. dicoccoides were susceptible, whilst in crosses between T. turgidum ssp. dicoccoides and durum wheat (AABB), F_1 plants expressed leaf rust resistance. In the F_2 progenies, resistant plants from the hexaploid crosses had fewer D chromosomes than the susceptible plants. Chromosomes 2B and 4B carried genes for leaf rust resistance whereas 1D and 3D carried suppressors. Suppressors of leaf rust resistance have also been located to the

A and B genomes. In crosses between *T. tauschii* and *T. aestivum*, Innes & Kerber (1994) identified genes for seedling resistance not previously identified. However, two of these genes did not express resistance in synthetic hexaploids but allowed the detection of three APR genes. The loss of seedling resistance in the hexaploids could be explained by the presence of suppressor genes on the A or B genomes (Kolmer, 1996).

The genetic basis of resistance conferred by lines in this study should also be determined. In studies examining the inheritance of slow rusting or PR to leaf rust in wheat (Lee & Shaner, 1985a; Lee & Shaner, 1985b; Broers & Jacobs, 1989; Jacobs & Broers, 1989; Singh, 1997), it has been found that two to three genes with small effects condition resistance. Although resistance controlled by several genes has greater potential for durability (Kolmer, 1996), combining them in a single genotype could be difficult.

Alien genes for rust resistance could be linked to unwanted yield or quality traits. Probably the best example of the introduction of undesirable genes alongside the transfer of disease resistance is the inferior bread-making quality of wheat lines carrying the 1BL/1RS wheat/rye chromosome translocation (Baum *et al.*, 1992). Although these lines have the *Sr31, Lr26, Yr9* and *Pm8* genes for resistance to stem, leaf and stripe rust, as well as powdery mildew, dough-stickiness has restricted their application in many wheat growing countries. Care should thus be taken not to introduce unwanted attributes linked to the genes controlling PR to leaf rust.

Due to practical limitations quantitative resistance components were studied in a few accessions only. Considering the present study and the data presented in Chapter 2, it could be assumed that valuable PR exists in this germplasm collection. Further detailed characterisation of macroscopic components may thus prove useful in identifying PR not detected previously.

Table 1. Infection types determined on flag leaves of *Triticum* accessions after inoculation with pathotype UVPrt13 of *Puccinia recondita* f. sp. *tritici*

Accession number	Accession[a]	Infection type[b]	
		Experiment 1	Experiment 2
1	Palmiet (*Triticum aestivum*)	3	3^+
2	*T. timopheevii* ssp. *araraticum* v. tumanianii (68)[c]	1	1
3	*T. turgidum* ssp. *dicoccum* v. rufum (356)	2^+	2
4	*T. turgidum* ssp. *anyleum* v. rufum (110)	2^{++}	2^+
5	*T. timopheevii* (324)	2^+	2^+
6	*T. turgidum* ssp. *dicoccoides* (317)	3^+	3
7	*T. turgidum* ssp. *carthlicum* v. stramineum (369)	1^{++}	1^{++}
8	*T. tauschii* (50)	1	1
9	*T. turgidum* ssp. *pyramidale* v. recognitum (I) (139)	2	2^+
10	*T. turgidum* ssp. *pyramidale* (141)	2^{++}	2^+
11	*T. turgidum* ssp. *pyramidale* v. recognitum (II) (138)	3	3^+
12	*T. turgidum* ssp. *pyramidale* v. compiticum (140)	3	3
13	*T. turgidum* ssp. *durum* v. obscurum (126)	1	1
14	*T. turgidum* ssp. *persicum* v. stramineum (132)	1^-	1^+

[a] Nomenclature according to the germplasm collection of the Department of Genetics, University of Stellenbosch, Stellenbosch, South Africa.
[b] Infection types were scored according to a 0 to 4 scale where 0=absence of macroscopic symptoms and 4=susceptibility. Plus or minus signs indicate variation above or below established pustule sizes.
[c] Numbers in parenthesis refer to the accession numbers used in Chapter 2.

Figure 1. Latent period of *Puccinia recondita* f. sp. *tritici* determined on flag leaves of Palmiet and *Triticum* accessions (listed in Table 1) after inoculation with pathotype UVPrt13. Data from the first experiment are given in (A) and from the second experiment in (B). Error bars represent positive standard deviations.
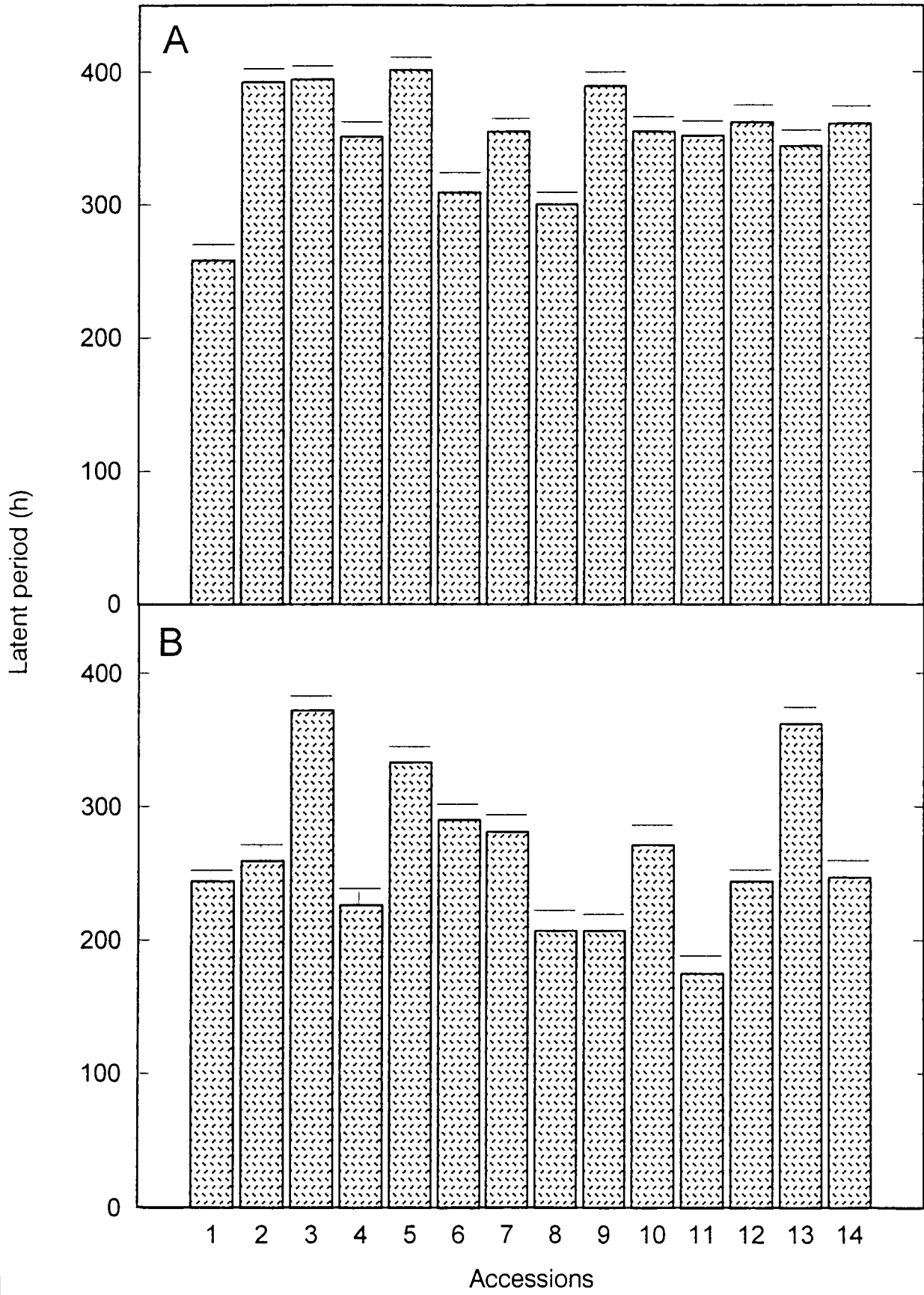
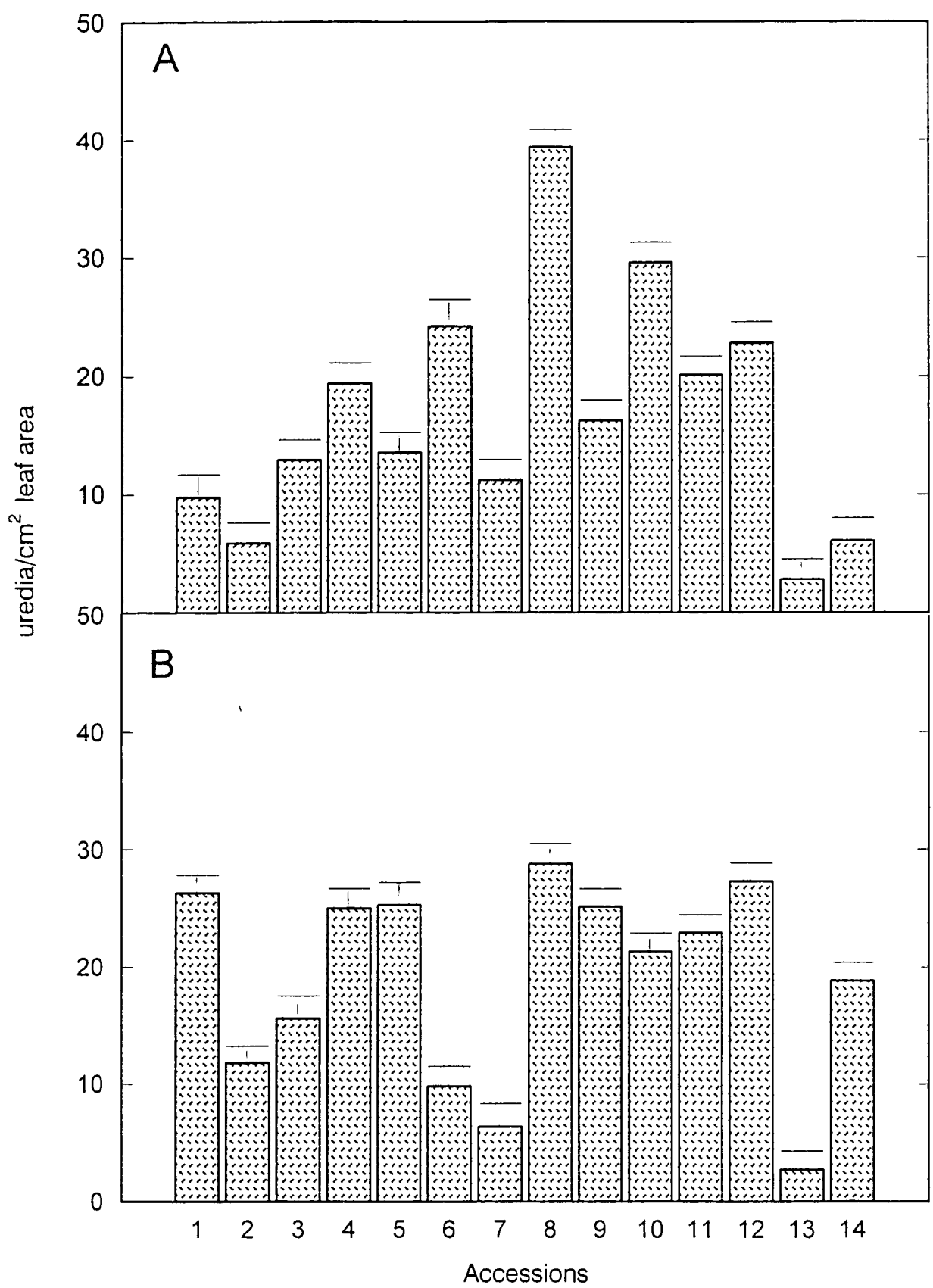Figure 2. Uredium density (number of uredia/cm^2 leaf area) determined on flag leaves of Palmiet and *Triticum* accessions (listed in Table 1) after inoculation with pathotype UVPrt13 of *Puccinia recondita* f. sp. *tritici*. Data from the first experiment are given in (A) and from the second experiment in (B). Error bars represent positive standard deviations.

Figure 3. Uredium size (mm^2) measured on flag leaves of Palmiet and *Triticum* accessions (listed in Table 1) after inoculation with pathotype UVPrt13 of *Puccinia recondita* f. sp. *tritici*. Data from the first and second experiments were pooled. Error bars represent positive standard deviations.
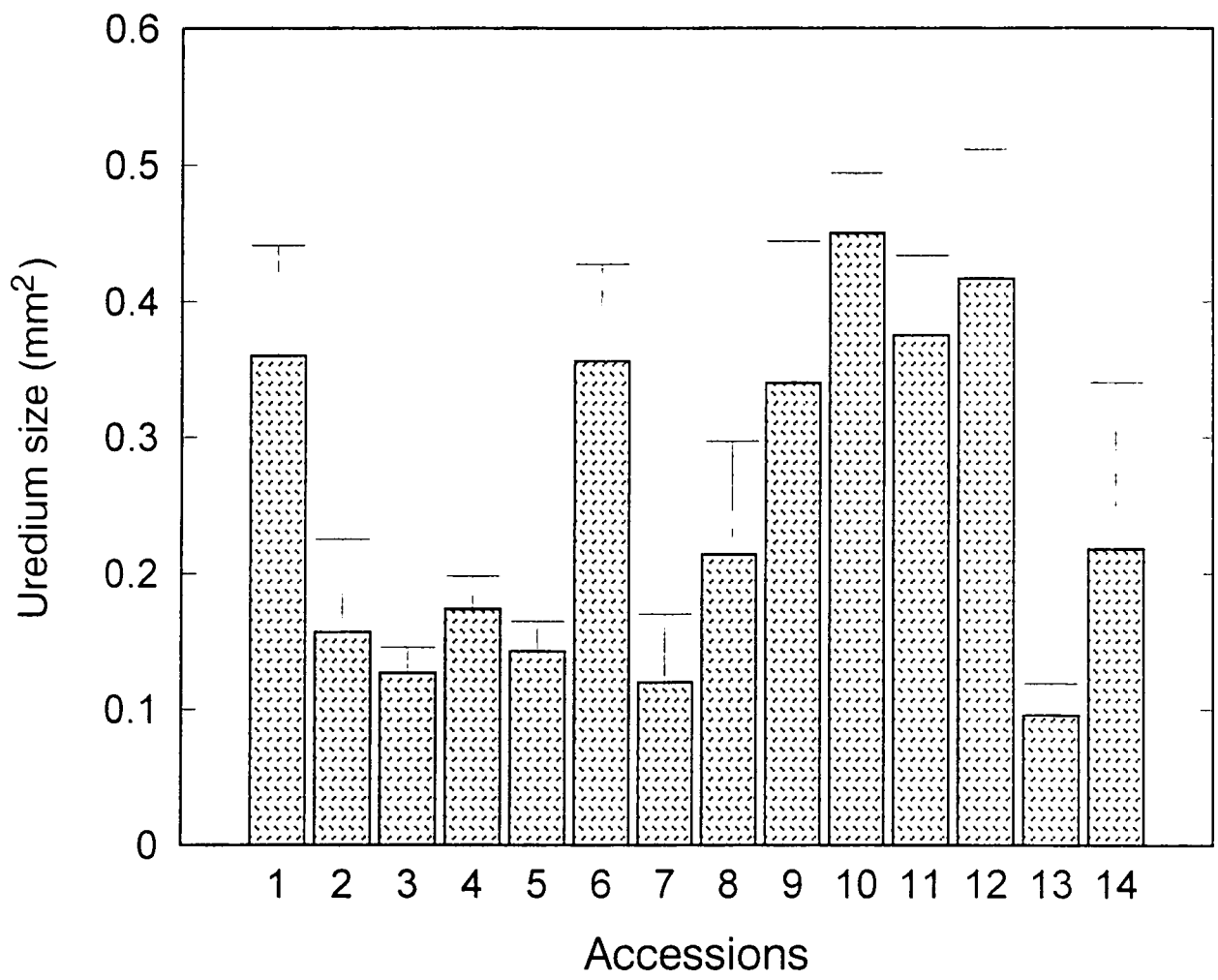
CHAPTER 4

HISTOPATHOLOGY OF RESISTANCE TO WHEAT LEAF RUST IN *TRITICUM TURGIDUM* AND *TRITICUM TIMOPHEEVII*

ABSTRACT

The histopathology of resistance to *Puccinia recondita* f. sp. *tritici* was investigated in selected *Triticum* accessions. Penetration and establishment of the wheat leaf rust pathogen were studied in flag leaves of *Triticum timopheevii*, *T. turgidum* ssp. *dicoccum*, *T. turgidum* ssp. *durum* and *T. turgidum* ssp. *compactum*. The *T. aestivum* wheats Thatcher (Tc) (susceptible common wheat control) and Tc*Lr19* (resistant common wheat control) were included in the experiment. Using fluorescence microscopy, infection sites of pathotype UVPrt13 were classified as either "prestomatal exclusion", "abortive penetration", "early abortion" or "colony formation". Nonpenetrating appressoria and aborted substomatal vesicles were regarded as abortive penetration. Flag leaf sections were prepared for phase-contrast microscopy by staining with either Trypan blue alone or in combination with a solution of picric acid in methyl salicylate. Leaf segments were screened for detection of infection sites at 100x whereas detailed obervations of haustoria and cell wall appositions, the latter visible as luminous structures, were conducted at 1000x (oil immersion). To confirm and expand light microscopy observations, upper and inner surfaces of epidermal tissue of *T. timopheevii* and *T. turgidum* ssp. *dicoccum* were fixed and prepared for scanning electron microscopy. Observations indicated that *T. timopheevii* expressed hypersensitive resistance typically associated with major genes, whereas *T. turgidum* ssp. *dicoccum*, *T. turgidum* ssp. *durum* and *T. turgidum* ssp. *compactum* showed varying degrees of prehaustorial resistance.

INTRODUCTION

Histological studies on interactions between plants and rust fungi have demonstrated that several mechanisms of resistance can be discerned (Heath, 1981a; Heath, 1982). In rust diseases prehaustorial resistance is one such defence mechanism assumed to be long lasting due to the absence of compatibility between the pathogen and the host

plant. Prehaustorial resistance implies that sporeling development is arrested before the formation of haustoria. Usually the sporelings develop normal haustorium mother cells, but a papilla is induced at the site of cell wall penetration. This type of resistance is very common in nonhost interactions (Heath, 1981a; Heath, 1982) and also in the presumably partial resistance of barley cultivars to *P. hordei* (Niks, 1982; Niks, 1983b). In posthaustorial resistance the fungus is arrested after the formation of at least one haustorium and, through a hypersensitive response, the cells that contain haustoria usually die. Race-specific, hypersensitive resistance to rust fungi has often been ephemeral, since the pathogen is able to develop races to which the resistance is not effective (Niks & Dekens, 1991). The presumably durable character of nonhost resistance and of partial resistance to barley leaf rust suggest that prehaustorial resistance may be difficult to overcome by rust fungi (Heath, 1981b; Heath, 1982; Heath, 1985).

The wild relatives of *T. aestivum* L. provide genetic variation for the improvement of disease resistance in bread wheat. Since certain *T. turgidum* L. and *T. timopheevii* (Zhuk.) Zhuk. accessions were rated as potentially valuable sources of resistance to *P. recondita* Rob. ex Desm. f. sp. *tritici* (Eriks. & Henn.) (Chapter 2), the objective of this study was to more precisely characterise resistance expression in these lines.

MATERIALS AND METHODS

Growing of *Triticum* species Three seeds each of Thatcher (leaf rust-susceptible control), TcLr19 (leaf rust-resistant control), *T. timopheevii*, *T. turgidum* ssp. *dicoccum* Schrank. *ex* Schübler, *T. turgidum* ssp. *durum* (Desf.) Husn. and *T. turgidum* ssp. *compactum* (Host) MacKey were planted in plastic pots containing 1 L of soil. Plants were grown in a leaf rust-free air-conditioned glasshouse cubicle. Natural daylight was supplemented with 120 μmol m^{-2}s^{-1} photosynthetically active radiation emitted by cool-white fluorescent tubes, arranged directly above plants, for 14 h each day. Once plants reached the one and a half leaf stage, 50 ml per pot of a 3g/L hydroponic nutrient solution (6.5:2.7:13 N:P:K plus micro-elements), were added three times weekly as a soil drench for the duration of the experiment. With the exception of scanning electron microscopy, the entire experiment was repeated in a similar study. During the first and

second experiments day/night temperature variation of $25.2 \pm 2.2°C/15.3 \pm 0.26°C$, and $24 \pm 1.54°C/14.5 \pm 0.43°C$, were recorded.

Inoculation and incubation Flag leaves were spray inoculated with freshly harvested spores of pathotype UVPrt13 of *P. recondita* f. sp. *tritici* suspended in distilled water containing Tween 20®. According to infection types produced on the South African differential set, this pathotype, which occurs commonly in South Africa, is avirulent to leaf rust resistance genes, *Lr3a, 3bg, 3ka, 11, 16, 20, 30* and virulent to *Lr1, 2a, 2b, 2c, 10, 14a, 15, 17, 24* and *26* (Pretorius *et al.*, 1995). The viability of urediospores was determined by microscopic examination of spore suspension droplets on 2% water agar plates incubated in the dark for 3 h at 20°C. A minimum of 99% urediospore germination was observed. After inoculation, plants were kept in the dark in a dew-simulation chamber at 19-22°C for 16 h. Upon removal from the chamber, plant surfaces were allowed to dry for 2 h in fan-circulated air before plants were transferred to a 6.5 m² air-conditioned glasshouse cubicle and maintained in conditions similar to those described for the pre-inoculation period.

Fluorescence microscopy

    Sample preparation and staining Two leaves per accession were sampled 88 h (h.p.i.) and 14 d (d.p.i.) post-inoculation and cut into 1 to 2-cm-long segments. Leaf segments were cleared and fixed in ethanol:dichloromethane (3:1 v/v) + 0.15% trichloroacetic acid for 24 h. Specimens were then washed twice in 50% ethanol for 15 min, twice for 15 min in 0.05 M sodium hydroxide and rinsed three times with water before being submerged in Tris[hydroxymethyl]aminomethane/hydrochloric acid (pH 5.8) and stained for 5 min in 0.1% diethanol (Uvitex 2B, Ciba-Geigy AG) (Niks & Dekens, 1991) in the preceding buffer. Following this, specimens were rinsed four times with water and washed with 25% aqueous glycerol for 30 min. Stained leaf sections were then stored in 50% glycerol containing a trace lactophenol to prevent deterioration of fungi and drying of material.

    Microscopic examination Leaf segments were used as whole mounts for fluorescence microscopy (Rohringer *et al.*, 1977; Kuck *et al.*, 1981). Observations were made at 100x or 400x, using a Nikon Optiphot epifluorescence microscope, on 20

randomly selected infection sites (IS) on each of five leaf segments. The filter combinations UV-1A (excitation filter 330-380 nm and barrier filter 420 nm) were used for fungal structures and B-2A (excitation filter 450-490 nm and barrier filter 520 nm) for autofluorescence measurements. Under these filter combinations all fungal structures except haustoria fluoresced a bright light-blue colour. Haustorium mother cells fluoresced extremely brightly. Host cells fluorescing an orange-yellow colour were considered necrotic, whereas unaffected healthy cells did not fluoresce (Rohringer *et al.*, 1977). Only infection sites where appressoria had formed over stomata were studied to determine the proportion of sites where substomatal vesicles, infection hyphae and haustorium mother cells occurred. The percentage prestomatal exclusion were calculated as the proportion of germtubes not producing any appressoria and appressoria not forming over stomatal openings. Abortive penetration (AP), defined by Parlevliet and Kievit (1986) as infection sites where appressoria did not penetrate a stomatal opening, or where infection structure development failed to proceed beyond the formation of substomatal vesicles (Niks, 1987) was determined. To obtain information on the mechanism of aborted penetration, the relative proportions of nonpenetrating appressoria and aborted substomatal vesicles were noted.

The number of infection sites displaying early abortion of infection structures was counted. Infection sites where six or less haustorium mother cells (HMC's) developed, were recorded as early abortions (Niks, 1983a). The number of early aborted infection sites with or without host cell necrosis (HCN) was also recorded. Infection sites culminating in colonies (more than six haustorium mother cells) were quantified, either with or without HCN. Colonies were then differentiated as either sporulating or nonsporulating. The number of HMC's was counted at 100x and confirmed at 400x magnification, where necessary. Where more than 30 HMC's were encountered, no further counts were made due to lack of accuracy. Infection sites crowded with more than one spore or appressorium were ignored, as well as those between the edge of the leaf and the first vein.

Dimensions of colonies, and of HCN associated with colonies, were measured with a calibrated eyepiece micrometer and corresponding areas (mm^2) calculated according to the formula: π x length x width/4. A hypersensitivity index (HI) (Kloppers & Pretorius, 1995) was calculated for each accession by expressing the necrotic area

as a fraction of the colony size. Uredium formation was calculated on leaf sections sampled 14 d.p.i. as the percentage infection sites that successfully established sporulating colonies. Coalescing colonies were excluded from measurements.

Prestomatal exclusion, abortive penetration, colony size, necrotic area, HI and uredium formation were studied at 14 d.p.i. only, whereas colony formation and number of HMC's were investigated at both sampling times.

Phase contrast microscopy

Sample preparation and staining Two leaves each of Thatcher, Tc*Lr19*, *T. timopheevii* and *T. turgidum* ssp. *dicoccum* were sampled 88 h.p.i. and cut into 1 to 2-cm-long segments. Leaf segments were cleared and fixed in ethanol:dichloromethane (3:1 v/v) + 0.15% trichloroacetic acid for 24 h before being boiled for 5 min in a 0.03 % solution of Trypan blue in lactophenol:ethanol (1:2 v/v). Specimens were then cleared by immersing them for 24 h in a saturated solution of chloral hydrate (5:2 w/v) and storing in 50% glycerol with a trace of lactophenol. To study cell wall appositions, specimens were transferred through a series of 80% (30 min), 90% (30 min) and 100% (2 x 30 min) ethanol for dehydration. Thereafter they were stained with a saturated solution of picric acid in methyl salicylate for 5 min (Niks, 1986). Stained leaf segments were mounted with adaxial sides upwards in methyl salicylate under cover slips sealed with nail varnish to prevent rehydration.

Microscopic examination Leaf segments were screened for detection of infection sites at 100x. Detailed observations of haustoria, and cell wall appositions visible as luminous structures, were conducted at 1000x (oil immersion). All colonised stomata observed in three leaf sections were studied by counting the number of haustoria and bright cell wall appositions in contact with hyphal tips or HMC's.

Scanning electron microscopy

Sample preparation and staining To study the development of early infection structures, two leaves each of Thatcher, Tc*Lr19*, *T. timopheevii* and *T. turgidum* ssp. *dicoccum* were sampled 24 h.p.i. Leaves were cut into 5 mm^2 sections and fixed in 3% gluteraldehyde. Leaf pieces were washed twice in 0.05 M phosphate buffer and post-fixed in 2% osmium tetroxide. Both fixatives were dissolved in 0.05 M phosphate buffer

(pH 6.8-7.2). Specimens were then dehydrated through an ethanol series and critical-point-dried in a Polaron critical point dryer. To investigate fungal structures inside the leaves, the epidermis was stripped using the leaf fracture technique described by Hughes & Rijkenberg (1985). Those pieces used to examine germination and appressorium formation on the leaf surface remained intact. Epidermis sections were sputter coated with gold in a Bio-Rad SEM coating system. The specimens were viewed with a JEOL WINSEM JSM-6400 scanning microscope operating at 5 kV.

Microscopic examination The infection structures observed were classified into one of the following categories: (1) presence of germtubes with no appressoria formed; (2) nonstomatal appressoria; (3) appressoria formed over stomata; (4) non-penetrating appressoria; (5) substomatal vesicle initial formed within the substomatal chamber; (6) aborted substomatal vesicle initial; (7) substomatal vesicle formed; (8) aborted substomatal vesicle; (9) primary infection hyphae formed and (10) secondary infection hyphae formed. Secondary infection hyphae were classified as hyphae that developed after the first HMC had formed. At least 50 infection sites were examined for each accession.

Infection types Disease reactions were rated according to a 0 to 4 infection type scale (Roelfs, 1988b) 14 d.p.i. on flag leaves of plants.

Statistical analysis Analysis of variance was done with SOLO (BMDP Statistical Software Inc., Los Angeles, CA), using the procedure for a general linear model. Standard deviations were determined to compare means. For each component, data from the first and second experiments were combined if experiment-by-treatment interaction was not significant, and if experiments, according to analysis of variance, were similar.

RESULTS

Fluorescence microscopy

Prestomatal exclusion Results from the two experiments were significantly (P<0.05) different. In the first experiment (Fig. 1A), prestomatal exclusion ranged

between 11% of the infection sites on the susceptible control, Thatcher, and 34% on *T. turgidum* ssp. *dicoccum*, and between 17% on Thatcher and 50% on *T. turgidum* ssp. *compactum* in the second experiment (Fig. 1B). In both experiments, the failure of germtubes to produce appressoria was more common than nonstomatal appressoria (Tables 1 and 2).

Abortive penetration Abortive penetration was defined as NPA (Fig. 2), ASSV and aborted substomatal vesicle associated with necrosis (ASSVN). Results from the two experiments differed significantly (P<0.05). Most lines had significantly more aborted infection units compared to the susceptible control, Thatcher (Fig. 3). In general, the second experiment showed a higher percentage abortive penetration (57.43%) than the first experiment (53.7%). A higher percentage ASSV were associated with necrosis in the second than in the first experiment in all accessions, except Tc*Lr19*. Considering the components of abortive penetration, NPA and ASSVN was not as conspicuous as was the abortion of substomatal vesicles (Fig. 4).

Early abortion In the first experiment, EA of fungal structures occurred frequently in the leaf rust resistant line, Tc*Lr19* (36.26%), whereas only 2.2% EA were recorded in the susceptible control, Thatcher (Table 1). EA was more frequent in the first than in the second experiment (Tables 1 and 2). In both experiments no EA were recorded in *T. turgidum* ssp. *dicoccum* and *T. turgidum* ssp. *compactum*. In experiment two, Thatcher also supported no EA. In the first experiment, all EA in Thatcher and *T. timopheevii* and most EA in Tc*Lr19* were associated with a hypersensitive response. In the second experiment, all EA's in Tc*Lr19* and *T. timopheevii* expressed a hypersensitive reaction (Tables 1 and 2).

Formation of colonies The formation of colonies (Fig. 5) differed significantly (P<0.05) between the two experiments for both sampling times. In both experiments, the highest percentage of infection sites classified as colonies, were observed in Thatcher (Tables 1 and 2).

88 h.p.i. Colonies were observed in all accessions in the second experiment, whereas none was recorded in the first experiment (Table 1) in Tc*Lr19* and *T. turgidum* ssp. *durum*.

14 d.p.i. In both experiments, colonies not associated with necrosis were absent in *T. turgidum* ssp. *dicoccum*. In Tc*Lr19*, no colonies were observed in the second

experiment, whereas all colonies observed in the first experiment were associated with necrosis. Also, in the first experiment no colonies were recorded in *T. timopheevii* and *T. turgidum* ssp. *compactum* and in the second experiment in *T. turgidum* ssp. *durum*. A higher percentage colonies was observed in the first than in the second experiment (Tables 1 and 2).

Colonies with necrosis

<u>88 h.p.i.</u> The percentage colonies associated with necrosis (Fig. 5) differed significantly ($P<0.05$) between the two experiments. In general, more colonies were associated with HCN in the first (16.2%) than in the second experiment (8.3%) (Tables 1 and 2).

<u>14 d.p.i.</u> Due to the failure of infection sites to produce colonies in the second experiment, HCN could not be recorded and was therefore not analysed for variance. Only Thatcher, Tc*Lr19* and *T. timopheevii* produced a hypersensitive reaction in the first experiment, and in the second experiment HCN was observed only in *T. timopheevii* and *T. turgidum* ssp. *compactum* (Tables 1 and 2).

Number of haustorium mother cells Results in the two experiments differed significantly. Fewer HMC's developed in Tc*Lr19* and the *Triticum* accessions than in Thatcher (Table 3) at both sampling times.

Colony size Due to the failure of infection sites to produce colonies in most of the lines, colony size was not analysed for variance. Colony size was only determined for Thatcher (0.868 mm^2), Tc*Lr19* (0.008 mm^2), *T. timopheevii* (0.009 mm^2) and *T. turgidum* ssp. *durum* (0.014 mm^2) in the first experiment (Table 4). In the second experiment, colony size was determined only for Thatcher (0.335 mm^2) and *T. turgidum* ssp. *compactum* (0.043 mm^2).

Necrotic area Assessment of necrotic area (mm^2) was not analysed for variance due to zero values in the data set. In the first experiment necrotic area could be determined only in Tc*Lr19* and *T. timopheevii*. (Table 4) In Tc*Lr19*, necrotic areas observed were bigger than that in *T. timopheevii*. In the second experiment no necrotic areas could be determined.

Hypersensitivity index HI could be determined for Tc*Lr19* and *T. timopheevii* only. Data for this index therefore were not analysed for variance. In the first experiment HI was more severe in Tc*Lr19* (1.75) than in *T. timopheevii* (1.44).

Uredium formation Sporulating colonies were observed only in Thatcher and only at 14 d.p.i. Thus, uredium formation and size were not analysed for variance. Uredium size was determined only for Thatcher. Results obtained in the experiments showed that uredia were larger in the first experiment (0.127 mm^2) than in the second experiment (0.082 mm^2).

Phase contrast microscopy

Haustoria and papillae Haustoria (Fig. 6A) were observed only in Thatcher in both experiments. More haustoria were observed in the first (mean 3.4/infection site) than in the second (mean 2.5/infection site) experiment. A few encapsulated haustoria were observed in Thatcher and encapsulated infection pegs in Tc*Lr19*. In *T. timopheevii* and Tc*Lr19* no haustoria or cell wall appositions could be seen due to necrosis and cell collapse. In *T. turgidum* ssp. *dicoccum* only vesicles, hyphae, and, a few non-penetrating appressoria were observed (Fig. 6B).

Scanning electron microscopy

Germtubes without appressoria The number of urediospores that germinated, but failed to produce appressoria were recorded (Figs. 7A and 8). The highest number of germtubes without appressoria were recorded in Tc*Lr19*. A fewer germtubes not forming appressoria were observed in *T. timopheevii*. On the susceptible control, Thatcher, more germtubes without appressoria were observed than on *T. turgidum* ssp. *dicoccum*.

Nonstomatal appressoria Nonstomatal appressoria were not analysed for variance due to zero values in data set. Nonstomatal appressoria were observed in Thatcher (1.9%), *T. timopheevii* (2.3%) and Tc*Lr19* (5.0%).

Appressoria formed over stomata The highest percentage germtubes successfully locating stomata and forming appressoria were observed in *T. turgidum* ssp. *dicoccum* and the lowest number in Tc*Lr19* (Figs. 7B and 9).

Nonpenetrating appressoria Due to zero values in the data set, nonpenetrating appressoria (Figs. 7C and 10) were not analysed for variance. Nonpenetrating appressoria were observed in Thatcher (1.9%), *T. timopheevii* (7.9%) and *T. turgidum* ssp. *dicoccum* (4.3%).

Substomatal vesicle initiation The number of substomatal vesicle initials observed varied between 10.8% on *T. timopheevii* and 43% on Tc*Lr19* (Figs. 11A and 12). In Thatcher, 12.7% aborted substomatal vesicle initials were detected.

Substomatal vesicle formation The number of substomatal vesicle initials that elongated to form mature vesicles (Figs. 11B and 13) was recorded. Most substomatal vesicles were observed in *T. timopheevii* (44.2%) and Thatcher (38.5%). In Tc*Lr19*, *T. timopheevii* and *T. turgidum* ssp. *dicoccum* some aborted substomatal vesicles were observed. Enumeration of structures indicated 93.75±8.84, 97.9±2.97 and 97.05±4.17 substomatal vesicles appeared normal in the latter three accessions.

Primary infection hyphae The percentage infection sites at which a primary infection hypha developed from one end of the substomatal vesicle (Fig. 14) were recorded. Primary infection hyphae were observed in all four accessions, with the highest percentage observed in *T. turgidum* ssp. *dicoccum*.

HMC formed on tips of primary infection hyphae The number of primary infection hyphae producing HMC's (Fig. 11C) is presented in Fig. 15. More than 60% primary hyphae formed HMC's in Tc*Lr19* in contrast with 26% in *T. turgidum* ssp. *dicoccum*.

Secondary infection hyphae No secondary infection hyphae were observed in *T. turgidum* ssp. *dicoccum* (Fig. 16). Most secondary infection hyphae were observed in Thatcher.

Infection types Low infection types were observed in Tc*Lr19* and the *Triticum* accessions, whereas Thatcher produced a susceptible infection type (Table 5).

DISCUSSION

To provide an overall view of the light microscopy observations, the relative proportions of prestomatal exclusion, abortive penetration, early abortion of infection structures, as well as the percentage of infection sites classified as colonies, are summarised in Fig. 17. According to these parameters, the *Triticum* species showed high levels of resistance when compared to Thatcher. More prestomatal exclusion, abortive penetration and EA were observed in Tc*Lr19* than in Thatcher. As was expected from

the susceptible control, Thatcher produced more colonies than Tc*Lr19*. In both experiments, a higher percentage prestomatal exclusion and AP were encountered in the accessions tested than in Thatcher. No colonies were detected in *T. turgidum* ssp. *dicoccum*. However, colonisation in *T. timopheevii* appeared more extensive than in Tc*Lr19*. In Tc*Lr19* more colonies aborted early than in the species.

Differences were observed between experiments and between leaves within the two experiments. Jacobs (1989a) mentioned a delicate balance between abortion and establishment which could be due to differences in experimental procedures (Niks, 1986, Jacobs, 1989d). In the vicinity of aborted infection structures in seedlings of the leaf rust-susceptible genotype Kaspar, yellow autofluorescence was present in the first experiment but not in the second experiment. This indicates the action of a gene for hypersensitive resistance with an expression influenced by environmental conditions. Having used bulked seed samples of *Triticum* species not necessarily selected for homogeneity, it is also possible that within-accession variation influenced results.

Spore germination and appressorium formation in all four accessions tested did not seem to be affected by resistance. In *T. turgidum* ssp. *dicoccum* and *T. turgidum* ssp. *durum* few nonstomatal appressoria were observed with fluorescence and electron microscopy techniques. In this study, quantitative analysis indicated that no nonstomatal appressoria occurred on *T. turgidum* ssp. *compactum* and *T. timopheevii*. This agrees with results obtained by various researchers. Niks (1981, 1982) investigated the possibility that, on partially resistant barley (*Hordeum vulgare*) genotypes, spore germination, appressorium formation, and/or stoma penetration may be affected. According to Niks (1981, 1982) no evidence for defence mechanisms in these early stages of the infection process was found. Even on a nonhost species (*T. aestivum*), appressorium formation (Niks, 1981) and stoma penetration (Niks, 1983a) by *P. hordei* were as successful as on barley. He also indicated that nonpenetration and SSV abortion did not play a significant role in nonhost resistance of adult plants (Niks, 1987). Jacobs (1989c) studied germination and appressorium formation of wheat leaf rust in barley (nonhost), wheat and species related to wheat *(T. dicoccum, T.dicoccoides, Ae. squarrosa* and *T. boeoticum*). Results obtained indicated no consistent differences in germination and appressorium formation between hosts and nonhosts. Also, genotypes with genes for partial or hypersensitive resistance to wheat

leaf rust do not seem to influence the pre-penetration phases (Jacobs, 1989a).

In this study, quantitative analysis in *T. turgidum* ssp. *compactum* indicated that nonpenetrating appressoria did not play a significant role. Furthermore, most IS did not display necrosis. *T. turgidum* ssp. *durum* had high levels of abortive penetration (± 73%) and a few nonpenetrating and nonstomatal appressoria were observed. With electron microscopy, collapsed SSV's were observed in Tc*Lr19*, *T. timopheevii* and *T. turgidum* ssp. *dicoccum*. Coutinho *et al.* (1993) observed SSV's in various stages of collapse at 48 h.p.i. in *Phaseolus vulgaris* infected with *Hemileia vastatrix*. Collapse of the infection structures occurred in nonhost tissue after the development of HMC's from the secondary infection hyphae (Coutinho *et al.*, 1993). Hu & Rijkenberg (1998) found no significant differences between the formation of early infection structures of *P. recondita* f. sp. *tritici* on and in susceptible and resistant wheat varieties. From their studies it was concluded that resistance expression is initiated after the formation of the first haustorium mother cells. This agrees with reports by Heath (1974, 1977) that the defence reaction to rust fungi in nonhost tissue usually commences after the first haustorium is formed.

Early abortion occurred in *T. turgidum* ssp. *durum* in the first experiment. In *T. timopheevii*, more EA was recorded than in Thatcher, but less than in Tc*Lr19*. No early aborted infection structures were observed in *T. turgidum* ssp. *dicoccum* and *T. turgidum* ssp. *compactum*. Although colonisation was observed at 88 h.p.i. in *T. turgidum* ssp. *dicoccum*, infection sites had no more than two HMC's, which indicated early abortion of structures. With phase contrast microscopy only hyphae, vesicles and on rare occasions, NPA were observed. Electron microscopy indicated collapsed vesicles and no secondary infection hyphae. Histological observations by Niks & Dekens (1987) showed that wheat and triticale exhibited a typical nonhost reaction to leaf rust of rye; sporelings of this fungus were arrested after the formation of primary infection hyphae and before the formation of extensively branched mycelium, mostly without necrosis of plant cells.

From results obtained by Lennox & Rijkenberg (1989) it appears that the formation of a successful haustorium from the primary infection hypha is a prerequisite for secondary infection hyphae formation. Heath (1974, 1977) working with nonhost interactions of maize, sunflower and cowpea rusts, found that, whether or not a

haustorium formed, secondary hyphae sometimes started to develop from the region of the infection hyphae adjacent to the HMC. These secondary infection hyphae remained short and never developed HMC's or haustoria of their own. According to Goodman & Novacky (1994), Ward, in 1902, mentioned a condition of incompatibilty in which no haustoria are formed, even though germination and penetration occur.

Very few, if any, cell wall appositions were observed in the *Triticum* accessions. Deposition of callose in association with the lack of haustoria does not seem to be common in nonhost reactions (Heath, 1977). Similarly, Jacobs (1989b) proved that prehaustorial exclusion by cell wall appositions is of minor importance in wheat partially resistant to *P. recondita* f. sp. *tritici*. The fact that the present study did not reveal cell wall appositions in the species tested, suggests the onset of other mechanisms of resistance, possibly the induction of pathogenesis-related proteins.

In Tc*Lr19*, EA's and ASSV's associated with necrosis were observed. This is in accordance with the fleck IT displayed. In *T. turgidum* ssp. *durum* (; IT) ASSV's associated with necrosis occurred infrequently. No EA's associated with necrosis were recorded in *T. turgidum* ssp. *dicoccum, T. turgidum* ssp. *durum* and *T. turgidum* ssp. *compactum*. All EA's in *T. timopheevii* were associated with necrosis, as well as a few ASSV's, which coincided with the resistance phenotype observed. In *T. timopheevii*, no observations were possible with phase-contrast microscopy due to extensive necrosis. This was due to either early or late hypersensitive resistance. Early hypersensitive resistance is characterised by a high percentage of early abortion, mainly with necrosis, a very low IT and, if present at all, small established colonies without sporogenic tissue. Late hypersensitive resistance, however, is characterised by relatively little early abortion and medium sized colonies with plant cell necrosis and a low to medium IT (Niks & Dekens, 1987). Jacobs *et al.* (1996) also indicated that early abortion of fungal structures in KS93U9, a leaf rust resistant bread wheat line, appears to have resulted from haustorium induced hypersensitive cell death inhibiting fungal development, rather than papilla formation. This provided evidence that the line exhibited posthaustorial resistance with necrosis. The lack of relationship between the growth of the rust colony and the occurrence of hypersensitive tissue also suggested that cell collapse is neither the only nor necessarily the most important factor in restricting the development of a rust colony in resistant varieties (Brown *et al.*, 1966).

Necrotic tissue in resistant hosts may therefore merely indicate that those hosts showing necrosis are more sensitive to the disturbances caused by the invading rust fungus than those that do not. Necrotic host cells may therefore be the consequence and not the cause of resistance, as is the limitation of mycelial growth (Brown *et al.*, 1966).

Results in this experiment clearly showed that resistance in *T. timopheevii* is typically hypersensitive and may thus not be durable. The prehaustorial resistance exhibited in *T. turgidum* ssp. *durum* and *T. turgidum* ssp. *compactum* may be valuable sources of nonhypersensitive resistance when transferred to cultivated wheat and should be further investigated and exploited.

Table 1. Components of resistance to pathotype UVPrt13 of *Puccinia recondita* f. sp. *tritici* in *Triticum* species as determined by fluorescence microscopy in the first experiment

Resistance component[a]	Sampling time[b]	Accessions					
		Thatcher[c]	TcLr19[d]	T. timopheevii (69)[e]	T. turgidum ssp. dicoccum (104)	T. turgidum ssp. durum (127)	T. turgidum ssp. compactum (143)
Prestomatal exclusion (%)							
No appressorium formed	14 d.p.i.	11.3±5.3	24.45±1.9	17.73±5.5	32.3±10.4	20.5±5.98	22.3±2.7
Nonstomatal appressorium	14 d.p.i.	0	0	0	1.5±0.8	1.6±1.96	0
Abortive penetration (%)							
Non-penetrating appressorium	14 d.p.i.	1.5±1.3	2±0.3	4.95±1.9	5.8±1.2	1.9±1.9	3.3±1.9
Aborted substomatal vesicle	14 d.p.i.	9.2±4.7	22.78±5.3	42.8±5.2	60.5±11.7	71.5±8.4	72.8±1.4
Aborted substomatal vesicle with necrosis	14 d.p.i.	0	6.78±0.4	14.12±3.8	0	0.6±0.5	1.34±0.4
Early abortion (%)							
Early abortion	14 d.p.i.	0	2.16±1.1	0	0	2±1.98	0
Early abortion with necrosis	14 d.p.i.	2.2±1.2	34.1±9.3	8.06±2.2	0	0	0
Colony formation (%)							
Colonies	88 h.p.i.	68.4±10.5	0	1.9±0.2	2.9±3.5	0	-[g]
	14 d.p.i.	31.76±12.8	0	0	0	1.9±1.01	0
Colonies with necrosis	88 h.p.i.	0	10.8±5.5	5.35±2.4	0	0	-
	14 d.p.i.	7.1±2.2	7.73±4.7	12.34±2.9	0	0	0
Sporulating colonies	14 d.p.i.	36.9±15.6	0	0	0	0	0

[a] All infection sites examined were classified as either prestomatal exclusion, aborted penetration, early abortion or colony formed. The relative proportions of subcomponents are shown.
[b] h.p.i. = hours post-inoculation; d.p.i. = days post-inoculation.
[c] Leaf rust-susceptible control.
[d] Leaf rust-resistant control.
[e] Numbers in parenthesis refer to the accession numbers used in Chapter 2.
[f] Means ± standard deviation.
[g] No data were obtained in the first experiment due to damage to leaves during inoculation.

Table 2. Components of resistance to pathotype UVPrt13 of *Puccinia recondita* f. sp. *tritici* in *Triticum* species as determined by fluorescence microscopy in the second experiment

Resistance component[a]	Sampling time[b]	Accessions					
		Thatcher[c]	Tc*Lr19*[d]	*T. timopheevii* (69)[e]	*T. turgidum* ssp. *dicoccum* (104)	*T. turgidum* ssp. *durum* (127)	*T. turgidum* ssp. *compactum* (143)
Prestomatal exclusion (%)							
No appressorium formed	14 d.p.i.	0	1.48±0.6	1.1±0.4	1.77±0.9	1.2±1.4	0
Nonstomatal appressorium	14 d.p.i.	0	1.48±0.6	1.1±0.4	1.77±0.9	1.2±1.4	0
Abortive penetration (%)							
Non-penetrating appressorium	14 d.p.i.	0	0.84±0.8	0	0	0.6±0.5	1.2±1.4
Aborted substomatal vesicle	14 d.p.i.	34±3.2	37.3±6.4	52.6±14.6	71.44±6.4	68.8±8	43.4±5.9
Aborted substomatal vesicle with necrosis	14 d.p.i.	1.4±0.6	9.4±4.5	16.49±8.7	0.9±0.1	2.1±2	6.6±1.3
Early abortion (%)							
Early abortion	14 d.p.i.	0	0	0	0	0	0
Early abortion with necrosis	14 d.p.i.	0	5.1±3.4	3.65±1.9	0	0	0
Colony formation (%)							
Colonies	88 h.p.i.	15.3±5.6	0.8±0.8	1.2±0.6	3.92±0.3	7.1±4.5	2.36±1.31
	14 d.p.i.	8.4±1.1	0	1.65±1.089	0	0	1.815±1.252
Colonies with necrosis	88 h.p.i.	2.6±1.5	2.96±1.3	2.7±0.96	0	0	0
	14 d.p.i.	0	0	1.64±0.8	0	0	1.2±1.3
Sporulating colonies	14 d.p.i.	39.4±2.2	0	0	0	0	0

[a] All infection sites examined were classified as either prestomatal exclusion, aborted penetration, early abortion or colony formed. The relative proportions of subcomponents are shown.
[b] h.p.i. = hours post-inoculation; d.p.i. = days post-inoculation.
[c] Leaf rust-susceptible control.
[d] Leaf rust-resistant control.
[e] Numbers in parenthesis refer to the accession numbers used in Chapter 2.
 Means ± standard deviation.

Table 3. Number[a] of haustorium mother cells of pathotype UVPrt13 of *Puccinia recondita* f. sp. *tritici* observed 88 hours post-inoculation and 14 days post-inoculation per infection site in flag leaves of adult Thatcher (leaf rust-susceptible), Tc*Lr19* (leaf rust-resistant) and *Triticum* accessions

Experiment	Sampling time[b]	Accessions					
		Thatcher	Tc*Lr19*	*T. timopheevii*	*T. turgidum* ssp. *dicoccum*	*T. turgidum* ssp. *durum*	*T. turgidum* ssp. *compactum*
First experiment							
	88 h.p.i.	16.43±5.65	3.11±2.42	4.65±2.13	1.89±0.6	0	-[c]
	14 d.p.i.	⟩30	7.67±0.71	12.47±4.85	0	14.17±7.19	0
Second experiment							
	88 h.p.i.	12.48±4.9	1.9±0.7	2.86±2.12	1.5±0.5	1.85±0.69	4.6±2.19
	14 d.p.i.	⟩30	0	0	0	0	27.75±4.5

[a] Means ± standard deviation.
[b] h.p.i. = hours post-inoculation; d.p.i. = days post-inoculation.
[c] No data were obtained in the first experiment due to damage to leaves during inoculation.

Table 4. Colony size[a] and size of the necrotic area produced by pathotype UVPrt13 of *Puccinia recondita* f. sp. *tritici* observed 14 days post-inoculation on flag leaves of adult Thatcher (leaf rust-susceptible), Tc*Lr19* (leaf rust-resistant) and *Triticum* accessions

Colony description	Accessions					
	Thatcher	Tc*Lr19*	*T. timopheevii*	*T. turgidum* ssp. *dicoccum*	*T. turgidum* ssp. *durum*	*T. turgidum* ssp. *compactum*
First experiment						
Colony size (mm^2)	0.868±0.021	0.008±0.001	0.009±0.001	0	0.014±0.005	0
Necrotic area (mm^2)	0	0.014±0	0.013±0.002	0	0	0
Second experiment						
Colony size (mm^2)	0.335±0.039	0	0	0	0	0.043±0.003
Necrotic area (mm^2)	0	0	0	0	0	0

[a] Means ± standard deviation.

Table 5. Infection types determined on the flag leaves of Thatcher (leaf rust-susceptible), Tc*Lr19* (leaf rust-resistant) and *Triticum* accessions after inoculation with pathotype UVPrt 13 of *Puccinia recondita* f. sp. *tritici*

Accession[a]	Infection type[b]	
	Experiment 1	Experiment 2
Thatcher (*Triticum aestivum*)	3[c]	2[++c]
Tc*Lr19*	0;	0;
T. timopheevii (69)[c]	;[cn]	;[c]
T. turgidum ssp. *dicoccum* (104)	0	0
T. turgidum ssp. *durum* (127)	0;	0;
T. turgidum ssp. *compactum* (143)	0	0

[a] Nomenclature according to the germplasm collection of the Department of Genetics, University of Stellenbosch, Stellenbosch, South Africa.
[b] Infection types were scored according to a 0 to 4 scale where 0=absence of macroscopic symptoms and 4=susceptibility. Flecks are indicated by ; and chlorosis and necrosis by "C" and "N", respectively. Plus or minus signs indicate variation above or below established pustule sizes.
[c] Numbers in parenthesis refer to the accession numbers used in Chapter 2.

Fig. 1. Percentage prestomatal exclusion of pathotype UVPrt13 of *Puccinia recondita* f. sp. *tritici* determined 14 d.p.i. on flag leaves of adult plants of the bread wheats (1) Thatcher (leaf rust-susceptible), (2) Tc*Lr19* (leaf rust-resistant) and the *Triticum* accessions, (3) *T. timopheevii*, (4) *T. turgidum* ssp. *dicoccum*, (5) *T. turgidum* ssp. *durum* and (6) *T. turgidum* ssp. *compactum* for the first (A) and the second (B) experiment. Observations were made with fluorescence microscopy. Error bars represent standard deviations.
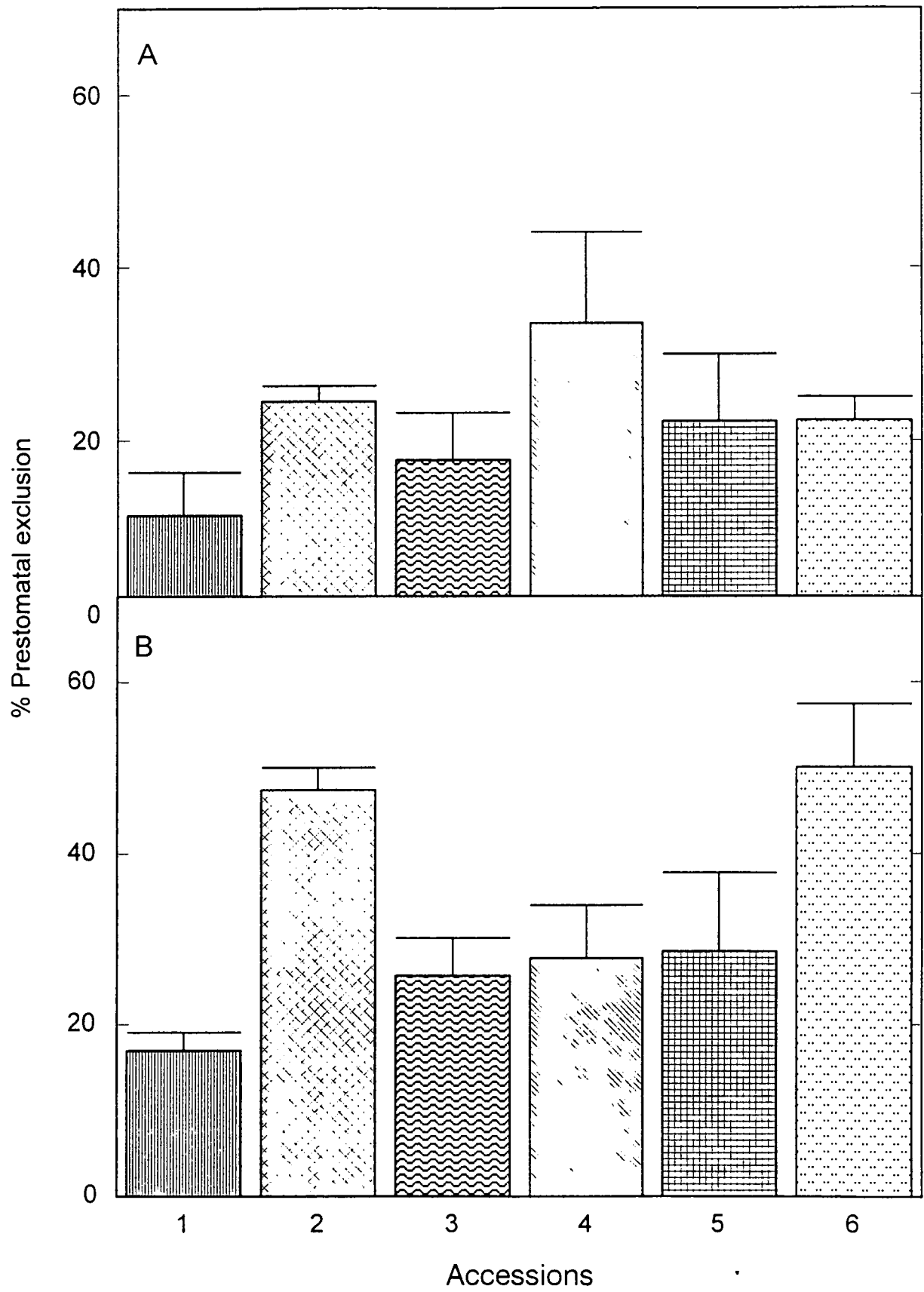
Fig. 2. Nonpenetrating appressorium of pathotype UVPrt13 of *Puccinia recondita* f. sp. *tritici* studied with fluorescence microscopy on *Triticum timopheevii*, 14 d.p.i. (400x). Abbreviations used: G = germtube; AP = appressorium; V = vesicle.   Scale bar represents 10 μm.

Fig. 3. Percentage abortive penetration of pathotype UVPrt13 of *Puccinia recondita* f. sp. *tritici* determined 14 d.p.i. on flag leaves of adult plants of the bread wheats (1) Thatcher (leaf rust-susceptible), (2) Tc*Lr19* (leaf rust-resistant) and the *Triticum* accessions, (3) *T. timopheevii*, (4) *T. turgidum* ssp. *dicoccum*, (5) *T. turgidum* ssp. *durum* and (6) *T. turgidum* ssp. *compactum* for the first (A) and the second (B) experiments. Observations were made with fluorescence microscopy. Error bars represent standard deviations.
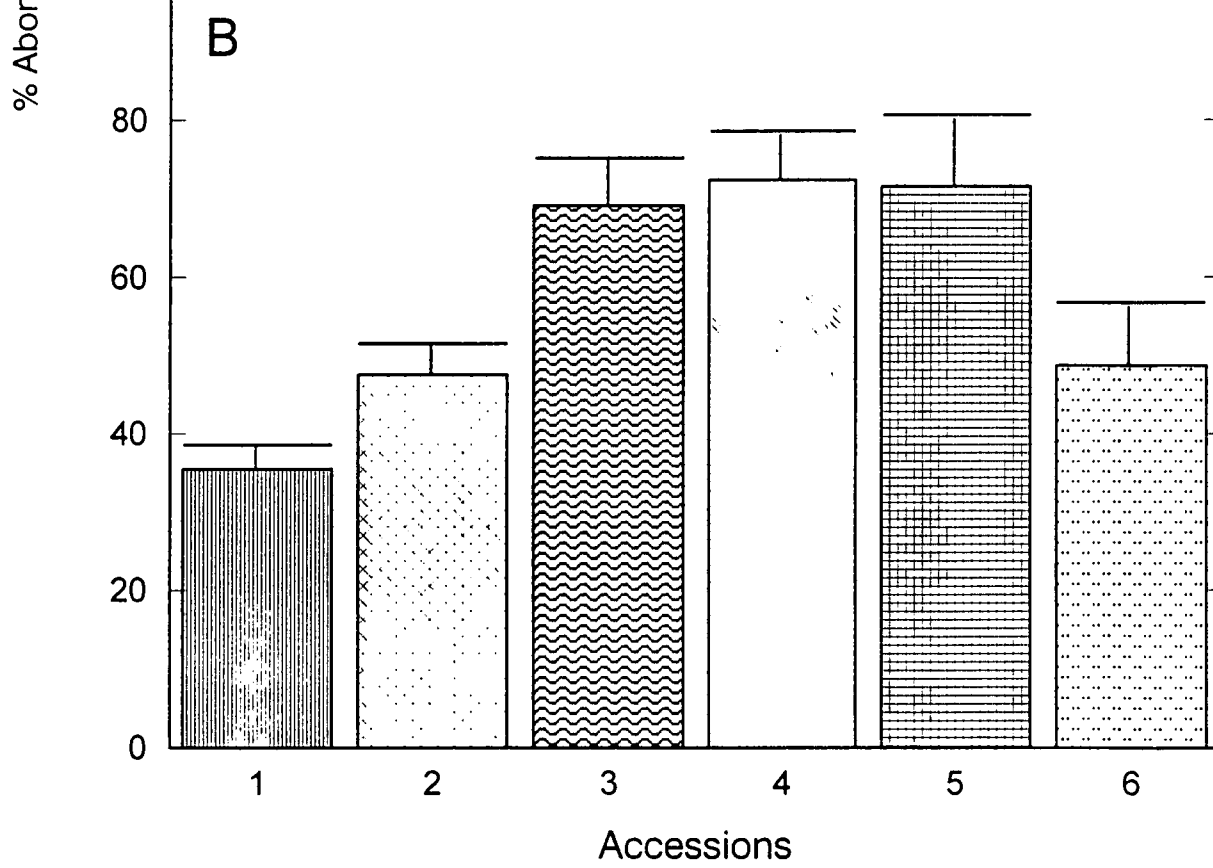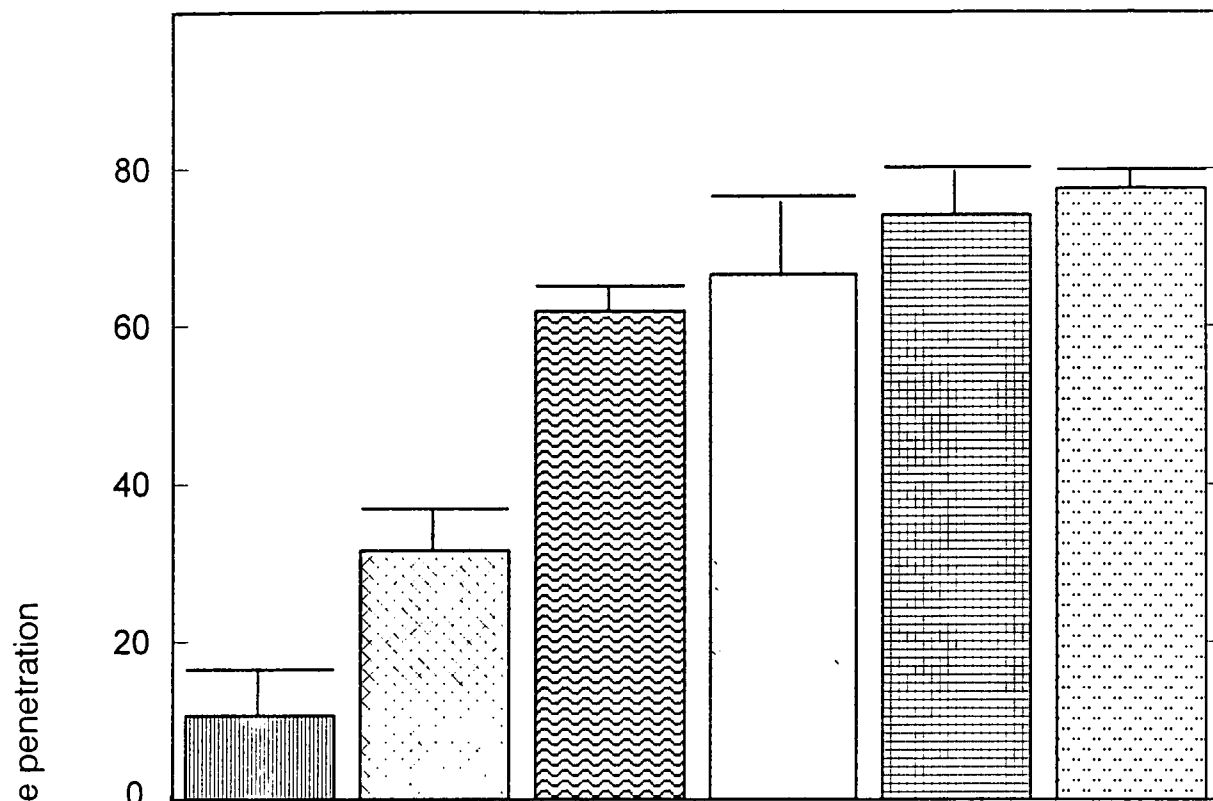
Fig. 4. The percentage infection sites of pathotype UVPrt13 of *Puccinia recondita* f. sp. *tritici*, determined 14 d.p.i. and classified as, respectively, nonpenetrating appressorium (NPA), aborted substomatal vesicle (ASSV) and aborted substomatal vesicle associated with necrosis (ASSVN) in the bread wheats (1) Thatcher (leaf rust-susceptible), (2) Tc*Lr19* (leaf rust-resistant) and the *Triticum* accessions, (3) *T. timopheevii*, (4) *T. turgidum* ssp. *dicoccum*, (5) *T. turgidum* ssp. *durum* and (6) *T. turgidum* ssp. *compactum* for the first (A) and the second (B) experiments. Observations were made with fluorescence microscopy.
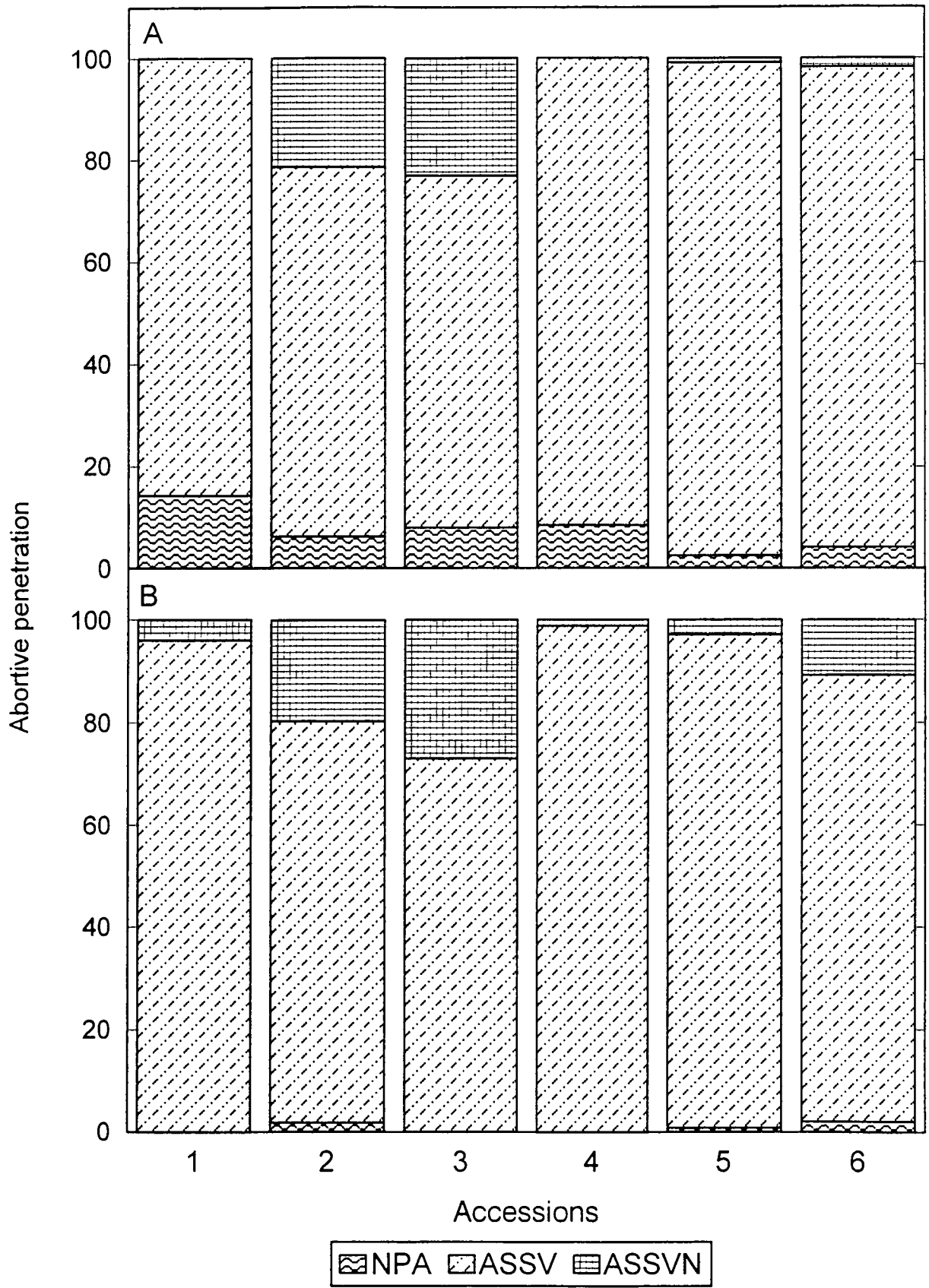
Fig. 5. A colony (A) of pathotype UVPrt13 of *Puccinia recondita* f. sp. *tritici* and associated host cell necrosis (B) at the same flag leaf infection site (14 d.p.i.) (400x) on *Triticum timopheevii*. Observations were made with fluorescence microscopy.
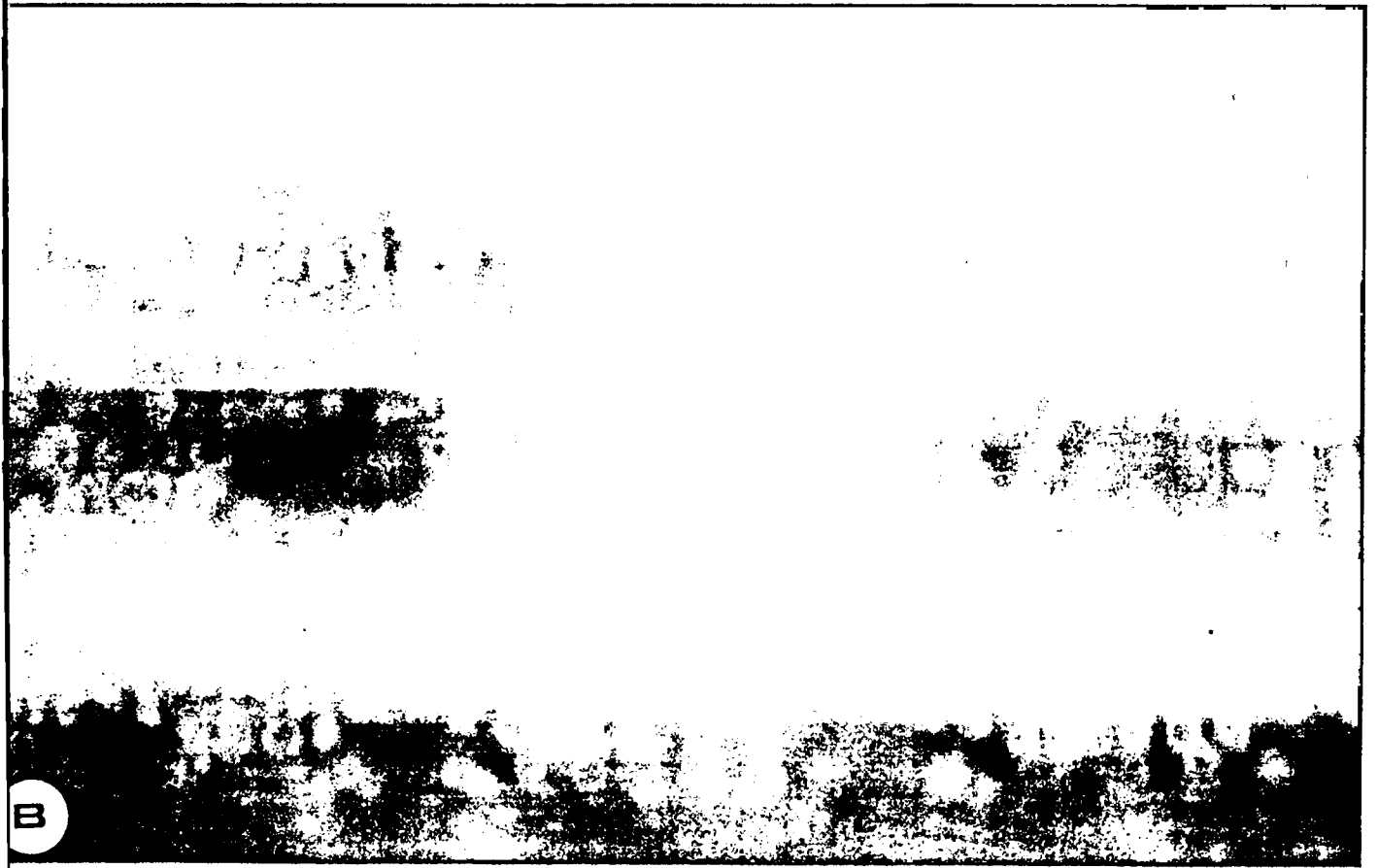
Fig. 6. Infection structures to pathotype UVPrt13 of *Puccinia recondita* f. sp. *tritici* studied with phase contrast microscopy. (A) haustorium and papillae in Thatcher (88 h.p.i.); (B) hypha in *Triticum turgidum* ssp. *dicoccum*. Abbreviations used: HMC = haustorium mother cell; HN = haustorium neck; H = haustorium; Hy = hypha; P = papilla. Scale bar represents 2.5 μm.
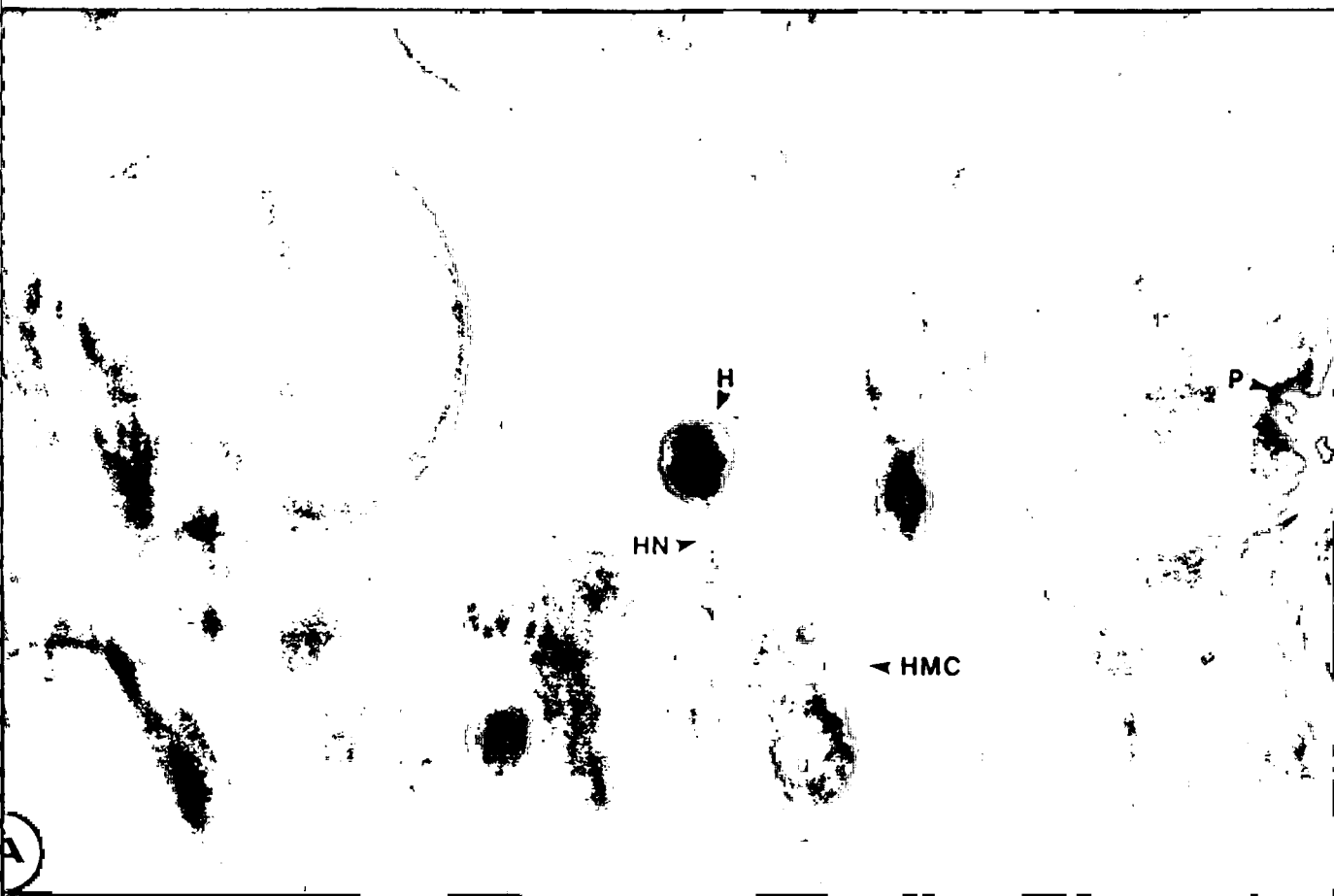
Fig. 7. Fungal structures studied, using scanning electron microscopy, to pathotype UVPrt13 of *Puccinia recondita* f. sp. *tritici*. (A) Germtube without an appressorium on Tc*Lr19*; (B) stomatal appressorium on *Triticum turgidum* ssp. *dicoccum*; (C) nonpenetrating appressorium on *T. timopheevii*. Abbreviations used: U = urediospore; G = germtube; AP = appressorium; St = stoma; V = vesicle. Scale bar represents 100 μm (A) and 10 μm (B & C).
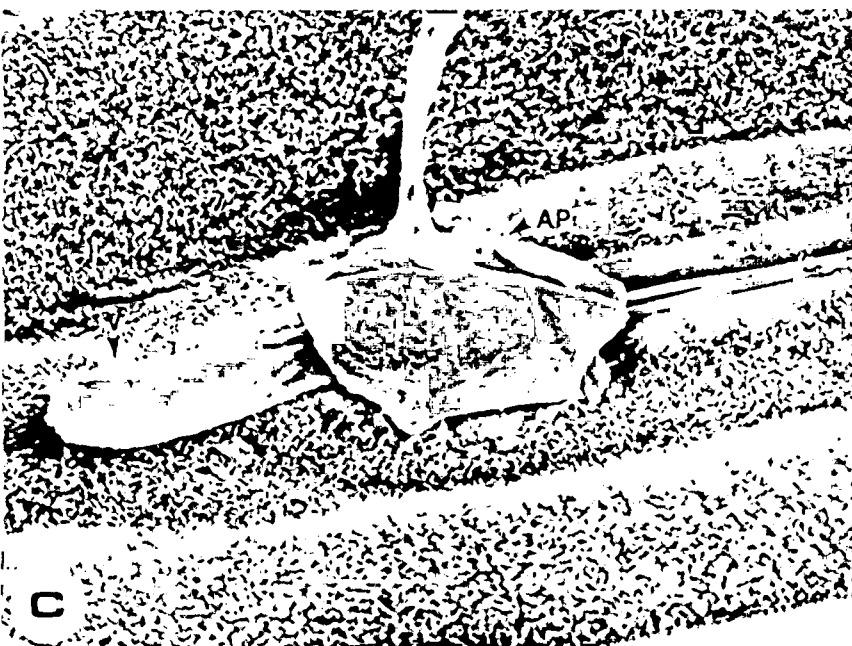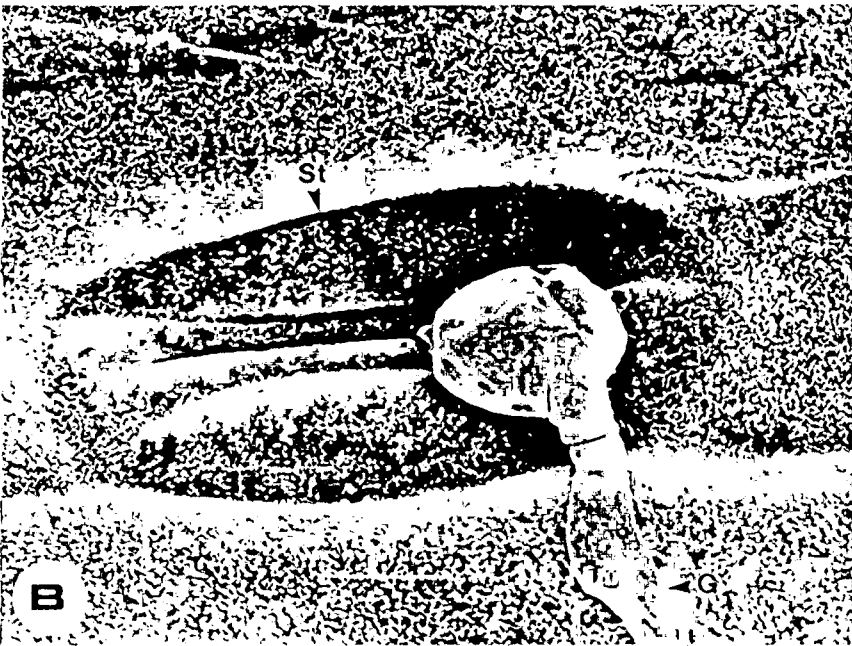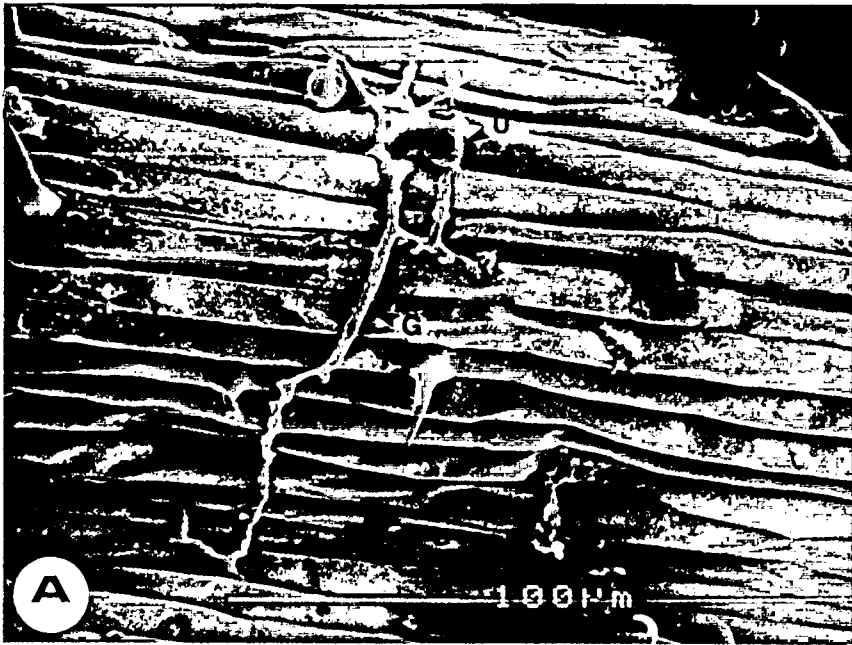
Fig. 8. Percentage germtubes of pathotype UVPrt13 of *Puccinia recondita* f. sp. *tritici* that did not form appressoria determined 24 h.p.i. on flag leaves of adult plants of the bread wheats (1) Thatcher (leaf rust-susceptible), (2) Tc*Lr19* (leaf rust-resistant) and the *Triticum* accessions, (3) *T. timopheevii* and (4) *T. turgidum* ssp. *dicoccum*. Observations were made with scanning electron microscopy. Error bars represent standard deviations.
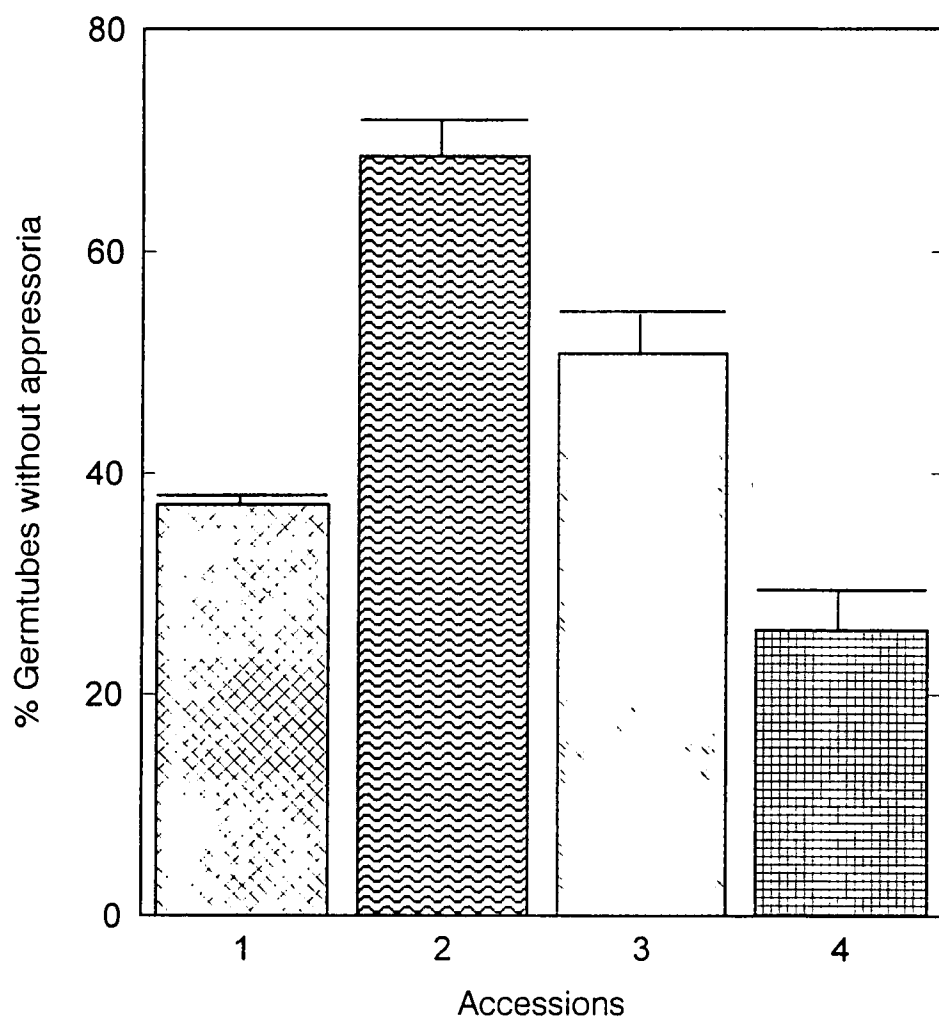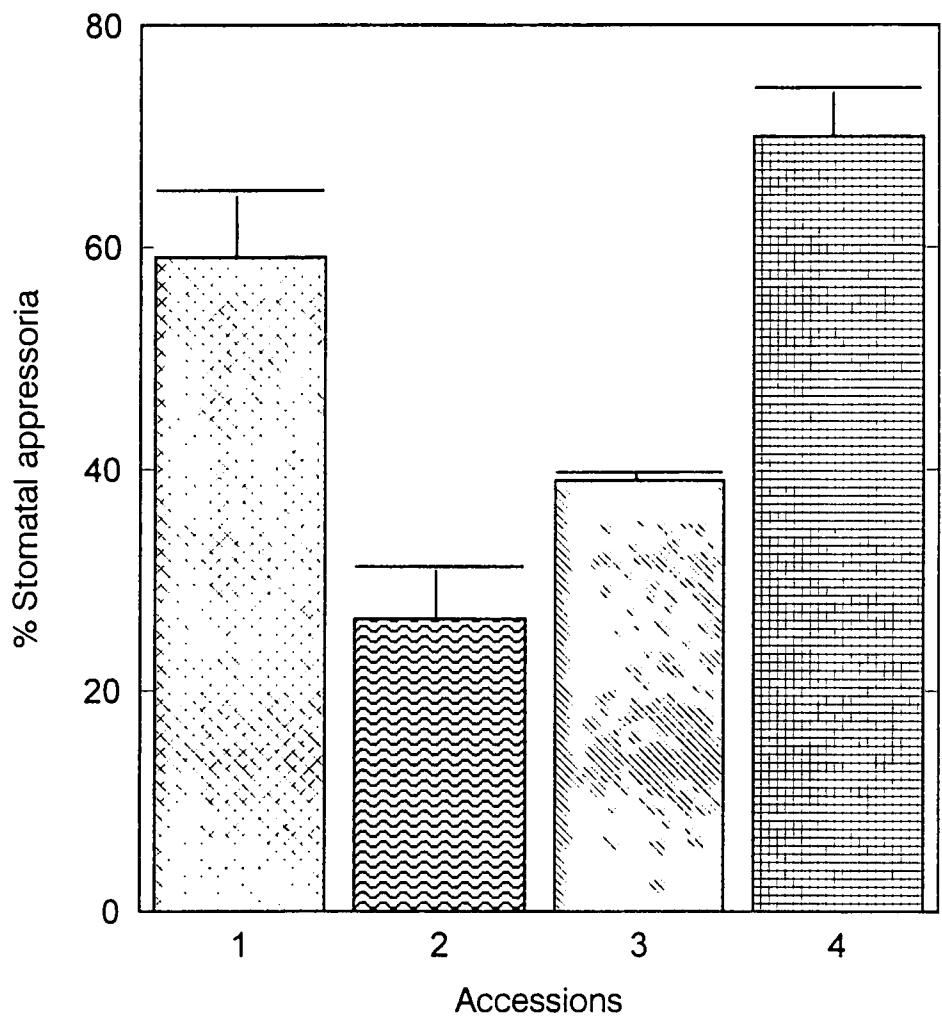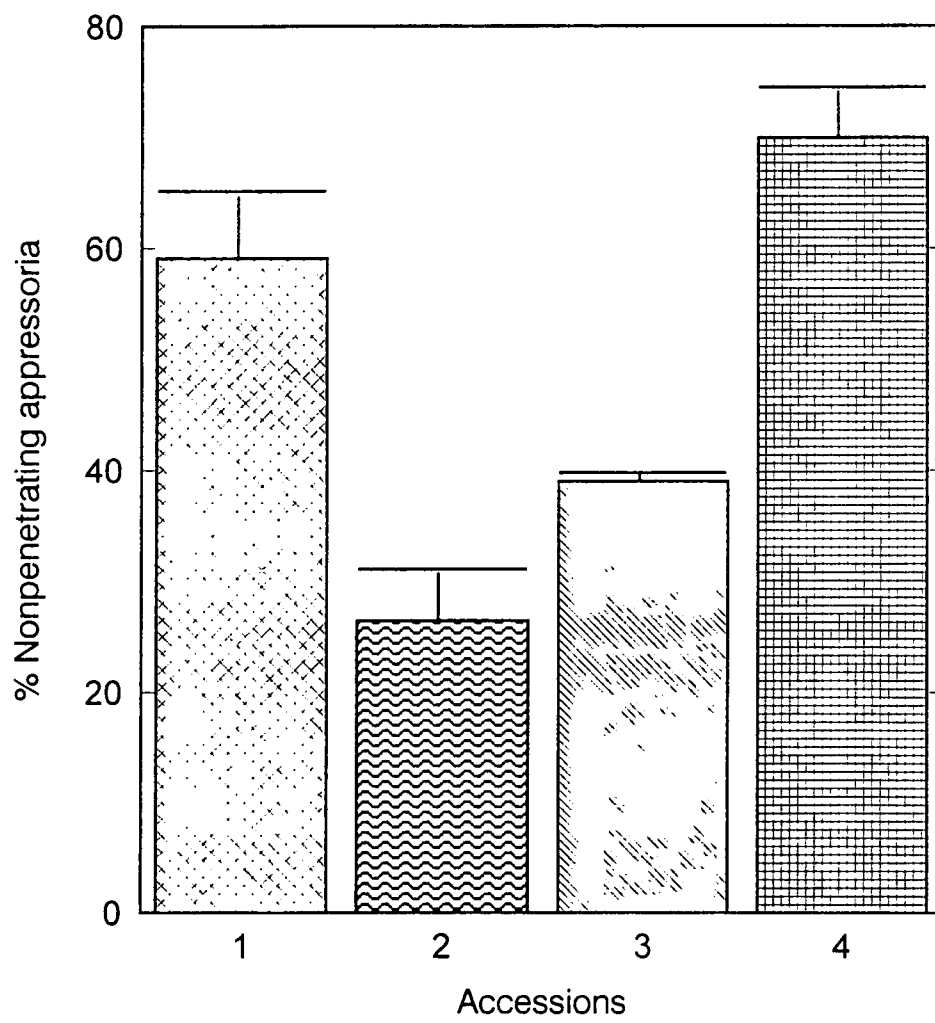
Fig. 9. Percentage appressoria of pathotype UVPrt13 of *Puccinia recondita* f. sp. *tritici* that formed over a stoma determined 24 h.p.i. on flag leaves of adult plants of the bread wheats (1) Thatcher (leaf rust-susceptible), (2) Tc*Lr19* (leaf rust-resistant) and the *Triticum* accessions, (3) *T. timopheevii* and (4) *T. turgidum* ssp. *dicoccum*. Observations were made with scanning electron microscopy. Error bars represent standard deviations.
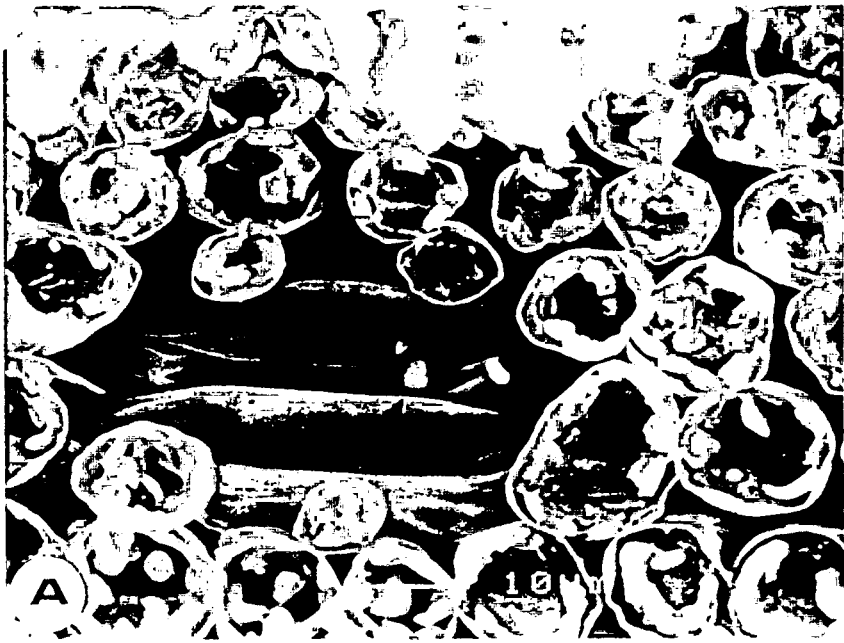
Fig. 10. Percentage nonpenetrating appressoria of pathotype UVPrt13 of *Puccinia recondita* f. sp. *tritici* determined 24 h.p.i. on flag leaves of adult plants of the bread wheats (1) Thatcher (leaf rust-susceptible), (2) Tc*Lr19* (leaf rust-resistant) and the *Triticum* accessions, (3) *T. timopheevii* and (4) *T. turgidum* ssp. *dicoccum*. Observations were made with scanning electron microscopy. Error bars represent standard deviations.
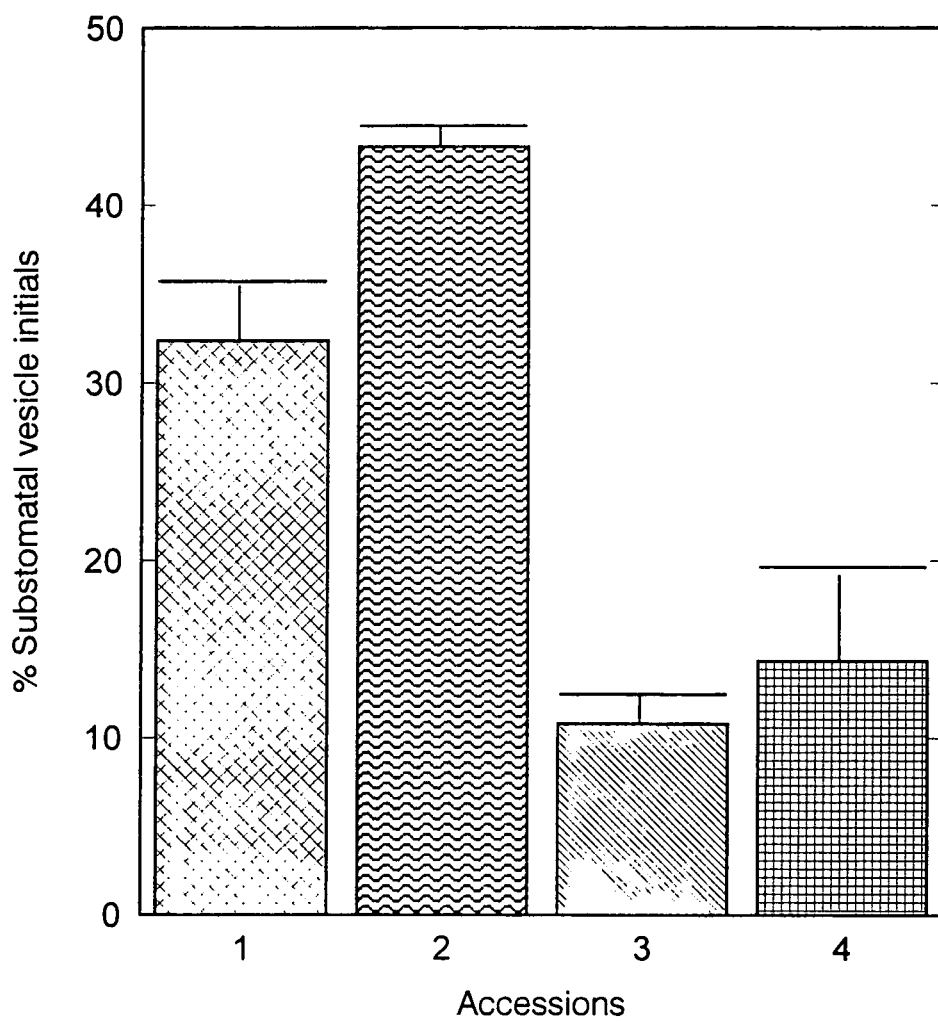
Fig. 11. Infection structures studied, using scanning electron microscopy, to pathotype UVPrt13 of *Puccinia recondita* f. sp. *tritici*. (A) Substomatal vesicle initial in Thatcher; (B) substomatal vesicle in *Triticum timopheevii*; (C) primary infection hypha with haustorium mother cell in Tc*Lr19*. Abbreviations used: HMC = haustorium mother cell; PIH = primary infection hypha; SSVI = substomatal vesicle initial; St = stoma; SSV = substomatal vesicle. Scale bar represents 10 μm.

Fig. 12. Percentage substomatal vesicle initials of pathotype UVPrt13 of *Puccinia recondita* f. sp. *tritici* determined 24 h.p.i. on flag leaves of adult plants of the bread wheats (1) Thatcher (leaf rust-susceptible), (2) Tc*Lr19* (leaf rust-resistant) and the *Triticum* accessions, (3) *T. timopheevii* and (4) *T. turgidum* ssp. *dicoccum*. Observations were made with scanning electron microscopy. Error bars represent standard deviations.
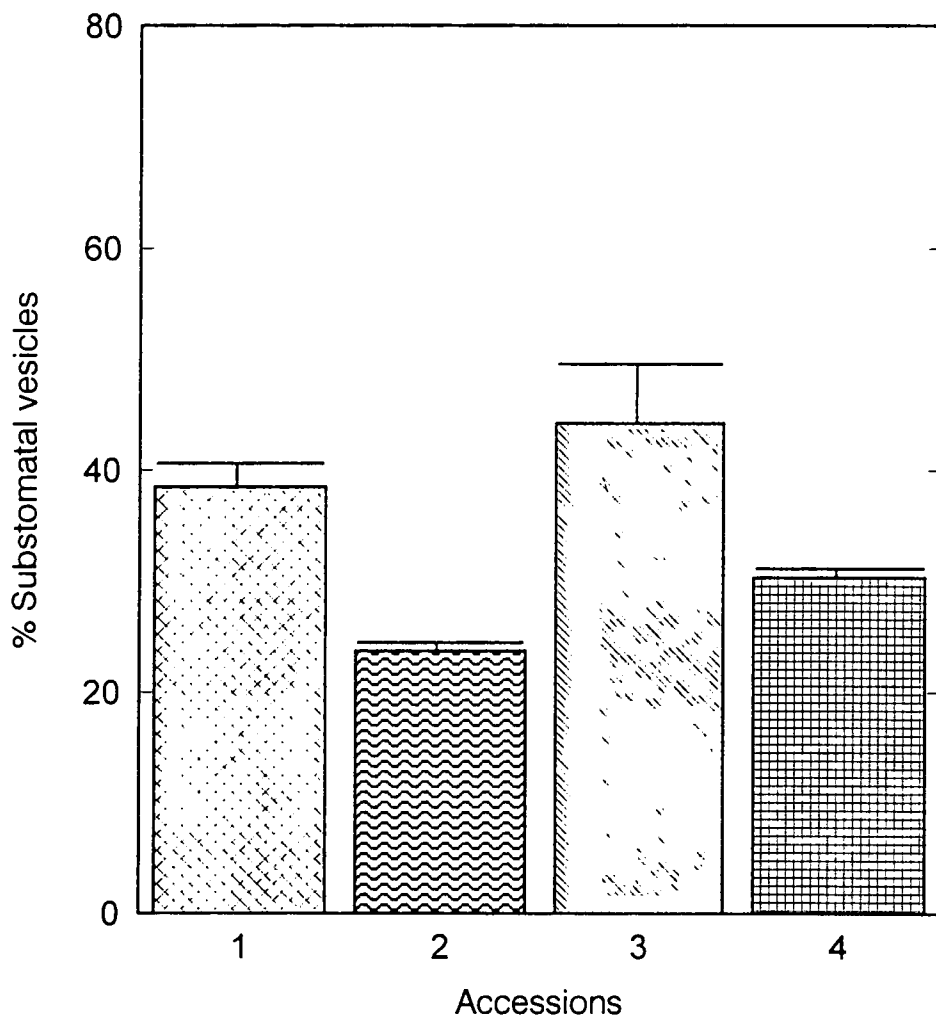
Fig. 13. Percentage substomatal vesicles of pathotype UVPrt13 of *Puccinia recondita* f. sp. *tritici* determined 24 h.p.i. on flag leaves of adult plants of the bread wheats (1) Thatcher (leaf rust-susceptible), (2) Tc*Lr19* (leaf rust-resistant) and the *Triticum* accessions, (3) *T. timopheevii* and (4) *T. turgidum* ssp. *dicoccum*. Observations were made with scanning electron microscopy. Error bars represent standard deviations.
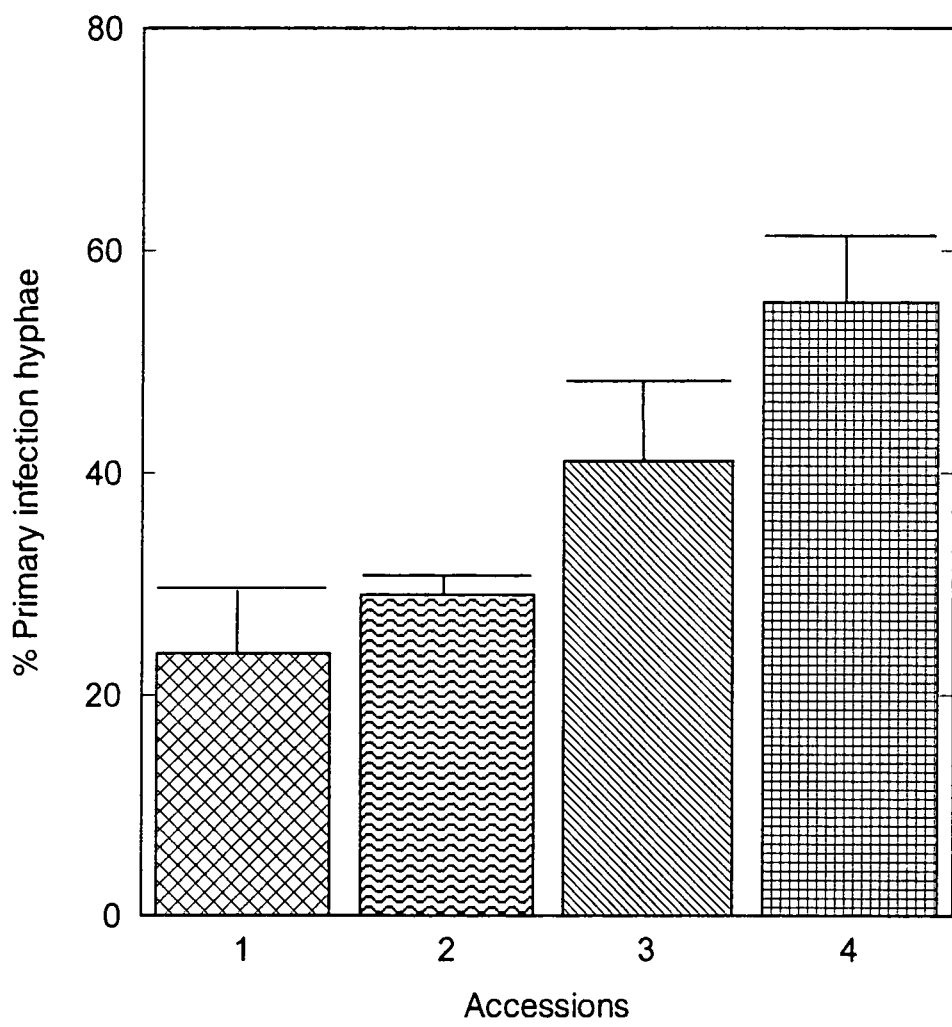
Fig. 14. Percentage primary infection hyphae of pathotype UVPrt13 of *Puccinia recondita* f. sp. *tritici* determined 24 h.p.i. on flag leaves of adult plants of the bread wheats (1) Thatcher (leaf rust-susceptible), (2) Tc*Lr19* (leaf rust-resistant) and the *Triticum* accessions, (3) *T. timopheevii* and (4) *T. turgidum* ssp. *dicoccum.* Observations were made with scanning electron microscopy. Error bars represent standard deviations.

Fig. 15. Percentage primary infection hyphae with a haustorium mother cell of pathotype UVPrt13 of *P. recondita* f. sp. *tritici* determined 24 h.p.i. on flag leaves of adult plants of the bread wheats (1) Thatcher (leaf rust-susceptible), (2) Tc*Lr19* (leaf rust-resistant) and the *Triticum* accessions, (3) *T. timopheevii* and (4) *T. turgidum* ssp. *dicoccum*. Observations were made with scanning electron microscopy. Error bars represent standard deviations.
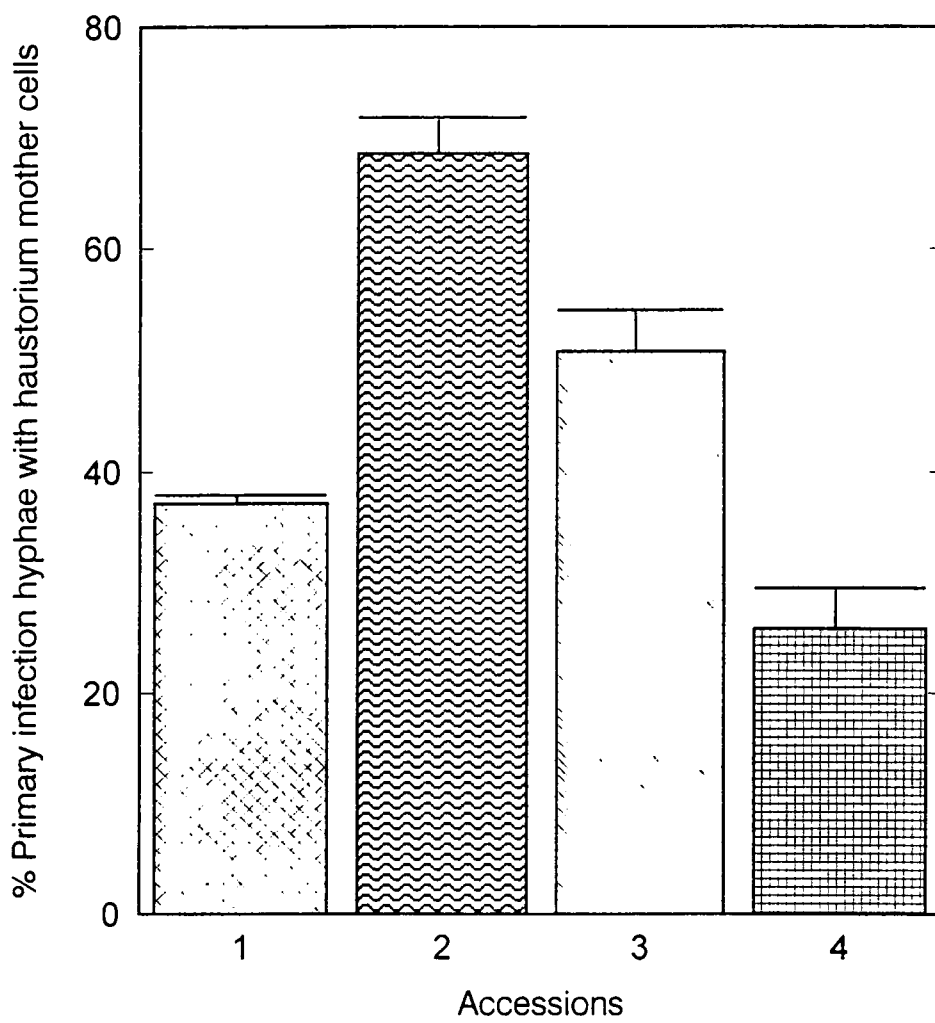
Fig. 16. Percentage secondary infection hyphae of pathotype UVPrt13 of *Puccinia recondita* f. sp. *tritici* determined 24 h.p.i. on flag leaves of adult plants of the bread wheats (1) Thatcher (leaf rust-susceptible), (2) Tc*Lr19* (leaf rust-resistant) and the *Triticum* accessions, (3) *T. timopheevii* and (4) *T. turgidum* ssp. *dicoccum*. Observations were made with scanning electron microscopy. Error bars represent standard deviations.
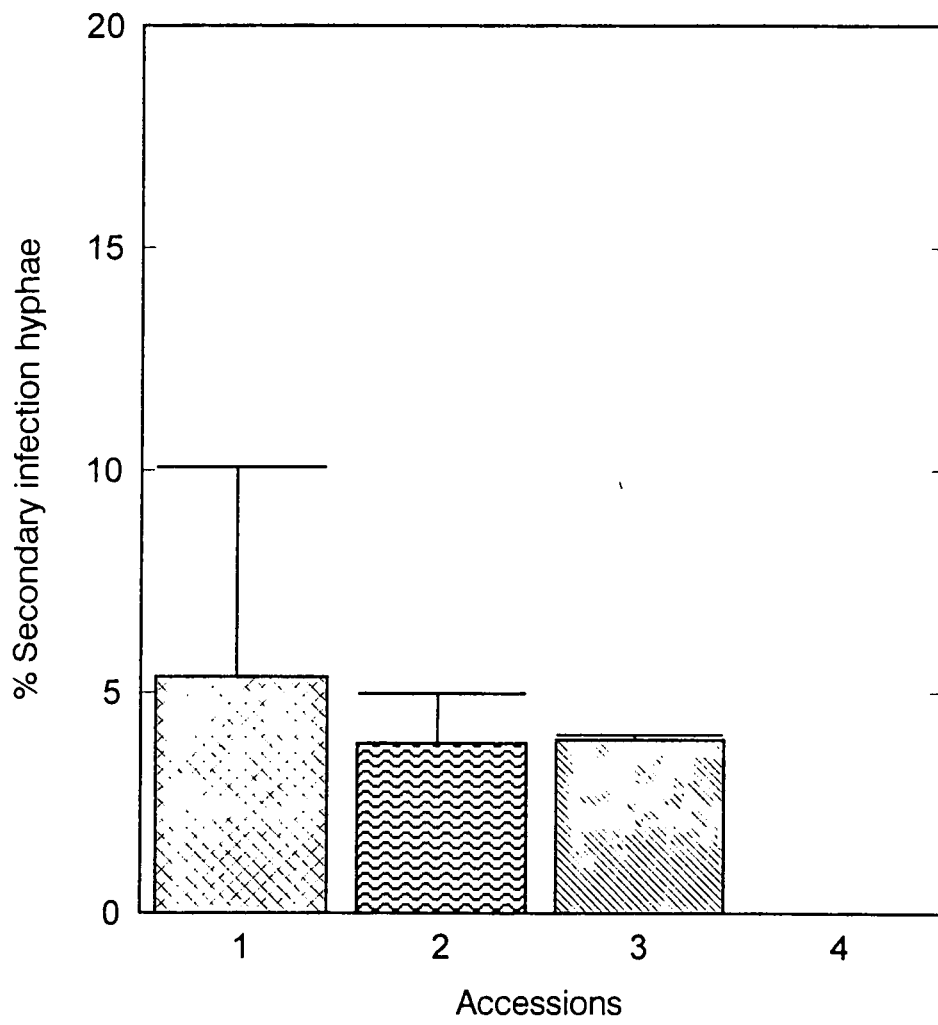
Fig. 17. The percentage infection sites of pathotype UVPrt13 of *Puccinia recondita* f. sp. *tritici* determined 14 d.p.i. and classified as, respectively, prestomatal exclusion, aborted penetration, early abortion and successfully established colonies in the bread wheats (1) Thatcher (leaf rust-susceptible), (2) Tc*Lr19* (leaf rust-resistant) and the *Triticum* accessions, (3) *T. timopheevii*, (4) *T. turgidum* ssp. *dicoccum*, (5) *T. turgidum* ssp. *durum* and (6) *T. turgidum* ssp. *compactum* for the first (A) and the second (B) experiments. Observations were made with fluorescence microscopy.
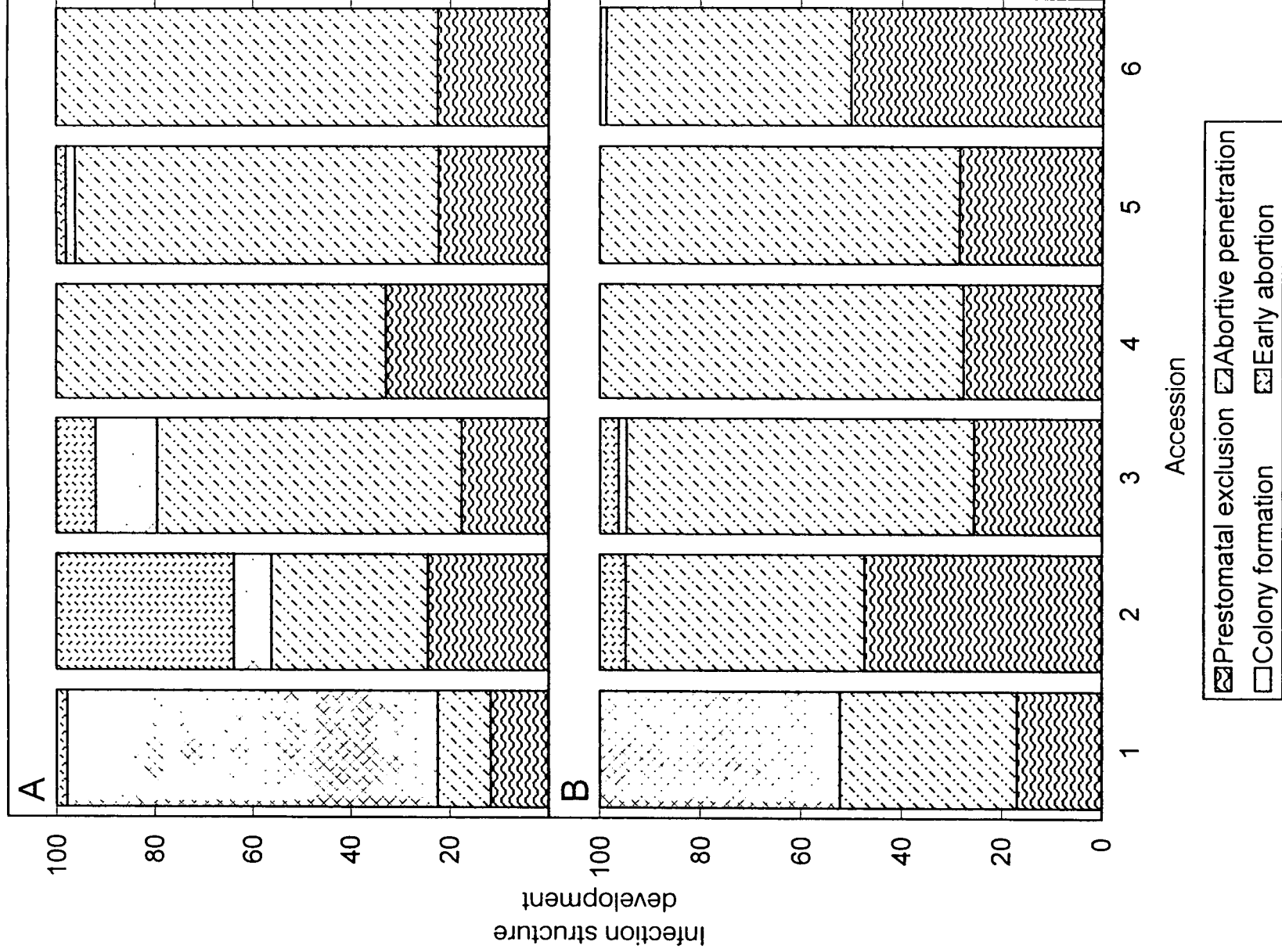
SUMMARY

The ability of rust pathogens to mutate and form new and virulent races, necessitates the broadening of the genetic base of resistance in common wheat to rust diseases. The wild relatives offer a rich reservoir of resistance genes. In an attempt to identify new sources of resistance to *Puccinia recondita* f. sp. *tritici*, 353 *Triticum* accessions, comprising diploid, tetraploid and hexaploid species were evaluated for seedling and adult-plant resistance to a mixture of pathotypes UVPrt2, 3, 9, and 13. In addition to infection type studies, plant height, growth habit and head type of adult plants were also recorded. One hundred and twenty six of the accessions were resistant to moderately resistant as seedlings to the pathotype mixture, whereas 180 were resistant or moderately resistant as adult plants. The number of days from planting to flag leaf stage varied from 54 to 187. High levels of resistance were observed in adult plants of *T. longissimum*, *T. sharonense*, *T. searsii* and *T. turgidum* ssp. *compactum*. *Triticum kotschyi* and *T. ventricosum* expressed hypersensitive infection types. Partial resistance (small pustules without any apparent chlorosis), was observed in *T. turgidum* ssp. *durum*, *T. turgidum* ssp. *pyramidale* and *T. tauschii*. In *T. turgidum*, which comprised 14 subspecies and 272 accessions, approximately 44% of the adult plants were resistant to moderately resistant compared to 24% of the seedlings.

According to these results 13 accessions, producing smaller or fewer leaf rust pustules, without the characteristic chlorosis and necrosis associated with hypersensitive resistance, were selected. Adult plants were quantitatively inoculated with pathotype UVPrt13 of *P. recondita* f. sp. *tritici*. Palmiet, a bread wheat cultivar susceptible to UVPrt13, was included as a control. Latent period of leaf rust, uredium size and density, and infection type were determined in two experiments. In the first experiment latent period ranged from 309 h to 401 h compared to 258 h in the susceptible control, Palmiet. In the second experiment Palmiet had a latent period of 244 h whereas those in the *Triticum* accessions ranged between 175 h and 372 h. Most accessions supported more uredia per cm^2 flag leaf surface than Palmiet in the first, but not in the second experiment. However, pustules were significantly smaller on most of the lines. Based on these components, *T. timopheevii* ssp. *araraticum* v. tumanianii, *T. turgidum* ssp. *durum* v. obscurum, *and T. turgidum* ssp. *persicum* v. stramineum, showed high levels of partial resistance.

Triticum turgidum and *T. timopheevii* accessions rated as potentially valuable sources of resistance were selected for histological studies on mechanisms of resistance. Penetration and establishment of the leaf rust pathogen were studied in flag leaves of *T. timopheevii*, *T. turgidum* ssp. *dicoccum*, *T. turgidum* ssp. *durum* and *T. turgidum* ssp. *compactum*. The *T. aestivum* wheats Thatcher (Tc) (susceptible common wheat control) and Tc*Lr19* (resistant common wheat control) were included in the experiment. Using fluorescence microscopy, infection sites of pathotype UVPrt13 were examined for the percentage prestomatal exclusion (germtubes not forming appressoria and appressoria not forming over stomata), abortive penetration (nonpenetrating appressoria and aborted substomatal vesicles), early abortion (six or less haustorium mother cells per infection site) and infection sites successfully culminating in colonies. Flag leaf sections were prepared for phase-contrast microscopy by staining with either Trypan blue alone or in combination with a solution of picric acid in methyl salicylate. To confirm and expand light microscopy observations, upper and inner surfaces of epidermal tissue of *T. timopheevii* and *T. turgidum* ssp. *dicoccum* were fixed and prepared for scanning electron microscopy. Observations showed that resistance in *T. timopheevii* was typically hypersensitive and may thus not be durable. The prehaustorial resistance exhibited in *T. turgidum* ssp. *durum* and *T. turgidum* ssp. *compactum,* may be valuable sources of nonhypersensitive resistance when transferred to cultivated wheat.

OPSOMMING

Die vermoë van roespatogene om te muteer en nuwe, virulente rasse te vorm, het 'n voortdurende behoefte aan nuwe weerstandsbronne tot gevolg. Kiemplasma van wilde spesies verwant aan broodkoring is 'n potensiële bron van weerstandsgene. In 'n poging om nuwe weerstandsbronne teen *Puccinia recondita* f. sp. *tritici* te identifiseer, is 353 *Triticum* inskrywings vir weerstand geëvalueer. Die inskrywings het bestaan uit diploïede, tetraploïede en heksaploïede tipes en is getoets teen die patotipes UVPrt2, 3, 9 en 13. Benewens infeksietipe is planthoogte, groeiwyse en aartipe bepaal. Eenhonderd-ses-en-twintig inskrywings was matig-weerstandbiedend tot volkome weerstandbiedend as saailinge. Hierteenoor was 180 inskrywings matig-weerstandbiedend tot volkome weerstandbiedend in die volwasse stadium. Die aantal dae vanaf plant tot die vlagblaarstadium het gewissel tussen 54 en 187. Hoë vlakke van weerstand is gevind in *T. longissimum*, *T. sharonense*, *T. searsii* en *T. turgidum* ssp. *compactum*. *Triticum kotschyi* and *T. ventricosum* het hipersensitiewe infeksietipes getoon. Gedeeltelike weestand (klein puisies sonder chlorose) is gevind in *T. turgidum* ssp. *durum*, *T. turgidum* ssp. *pyramidale* en *T. tauschii*. In *T. turgidum*, bestaande uit 14 subspesies and 272 inskrywings, was ongeveer 44% volkome weerstandbiedend tot matig-weerstandbiedend as volwasse plante teenoor 24% in die saailingstadium.

Na aanleiding van bogenoemde resultate is 13 inskrywings geselekteer. Hierdie inskrywings het nie chlorotiese of nekrotiese reaksies, soos normaalweg geassosieër met hipersensitiwiteit, getoon nie. Volwasse plante is kwantitatief geïnokuleer met patotipe UVPrt13 van *P. recondita* f. sp. *tritici*. Palmiet is ingesluit as vatbare kontrole. Latente periode van blaarroes, urediumgrootte en -digtheid en infeksietipe is bepaal. Die eksperiment is in sy geheel herhaal. In die eerste eksperiment het latente periode in die inskrywings tussen 309 h en 401 h gevarieër, in teenstelling met 258 h in Palmiet. In die tweede eksperiment het Palmiet 'n latente periode van 244 h getoon. Latente periode in die inskrywings het gewissel tussen 175 h en 372 h. In meeste van die inskrywings is hoër urediumdigthede aangeteken as op Palmiet, hoewel puisies in die spesie-aanwinste meestal kleiner was. Gebaseer op bogenoemde resultate, beskik *T. timopheevii* ssp. *araraticum* v. tumanianii, *T. turgidum* ssp. *durum* v. obscurum, en *T. turgidum* ssp. *persicum* v. stramineum, oor hoë vlakke van gedeeltelike weerstand.

Weerstandsmeganismes is histologies bestudeer in inskrywings van *T. turgidum* en *T. timopheevii.* Penetrasie deur en vestiging van die patogeen is bestudeer in vlagblare van *T. timopheevii*, *T. turgidum* ssp. *dicoccum*, *T. turgidum* ssp. *durum* en *T. turgidum* ssp. *compactum.* Die broodkoring-kultivars Thatcher (vatbare kontrole) en Tc*Lr19* (weerstandbiedende kontrole) is ingesluit. Fluoressensie mikroskopie is gebruik om infeksiepunte te bestudeer en die persentasie prestomatale uitsluiting (kiembuise wat nie appressoria vorm nie, of nie-stomatale appressoria), abortiewe penetrasie (nie-penetrerende appressoria of abortiewe substomatale vesikels), vroeë abortering (ses of minder haustoriumoederselle per infeksiepunt) en infeksiepunte wat kolonies vorm, is genoteer. Blare is ook gekleur met trypanblou en 'n versadigde oplossing van pikriensuur in metielsalisilaat. Om die vorming van infeksiestrukture op en in blaarweefsel van *T. timopheevii* and *T. turgidum* ssp. *dicoccum* te bestudeer, is skandeer-elektronmikroskopie gebruik. *Triticum timopheevii* het 'n tipiese hipersensitiewe reaksie getoon en dié weerstand is moontlik nie volhoubaar nie. Die prehaustoriale weerstand in *T. turgidum* ssp. *durum* and *T. turgidum* ssp. *compactum* kan moontlik waardevolle bronne van nie-hipersensitiewe roesweerstand wees.

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